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Executive Director; EUNICE THOMAS MINER

SECTION OF BIOLOGICAL AND MEDICAL SCIENCES

THE GENESIS OF CANCER AND POSSIBLE INTERFERENCE WITH ITS GROWTH*

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The cancer problem necessitates research that leads into such various fields of science as anatomy and pathology, physiology and embryology, histology and cytology, botany and zoology and, further, into microbiology and virus research, genetics and immunology, biochemistry and biophysics, endocrinology and nutrition, pharmacology and toxicology, and biometry and statistics. Each is important to cancer research. To do justice to these various disciplines requires so much specialized study that one tends to lose sight of the cancer problem as a whole. The danger of the present situation is its extreme subdivision. As specialized work and the knowledge gained through it generally entail more specialization, the knowledge acquired through study of the individual processes has the peculiar result of becoming an obstacle to the study of the whole. I believe, therefore, that we must change our present way of thinking and that our most important task should be the study of the problem in its entirety. I have called attention to this situation in my book *Cancer - Nature, Cause and Cure* (1957).

I propose first to give a general summary of the present situation in cancer research, beginning with the established facts ascertained by research.

It is an established fact that: (1) the cancer problem is still unsolved; (2) there is as yet no remedy for cancer; (3) there is still no infallible diagnostic method for its early detection; (4) cancer in its early stage is painless, and pain does not occur until caused by displacement and infiltration; (5) experimental cancer can be produced at will; (6) cancer can develop from the action of the most diverse noxae, including those from radiation, or become superimposed on chronic conditions; (7) cancer is not a bacterial infection; (8) cancer cells are characterized by uninhibited growth; (9) cancer tissue, as compared to normal tissue, exhibits quantitatively altered biochemical characteristics; (10) cancer cells are less differentiated than normal cells; (11) any cell in the organism can become the mother cell of a malignant tumor; (12) primary cancer can produce metastatic tumors; (13) cancer is the most frequent cause of death in highly developed countries (if we exclude death due to wear and cardiovascular diseases); and (14) the aim of all cancer therapy is to remove or destroy the cancer cell or to arrest its growth either by surgery, irradiation, or tumor-inhibiting chemical agents.

* This paper, illustrated with slides, was presented at a meeting of the Section on November 9, 1959.

I now state the controversial points. It is controversial whether: (1) cancer develops as a result of mutation; (2) cancer is hereditary; (3) cancer arises because of a delayed development of embryonic cells, that is, whether cancer cells are in fact embryonic cells; (4) cancer is caused by a virus; (5) cancer develops as a result of an endocrine disorder; (6) cancer develops as a result of malnutrition; (7) cancer cells develop as a result of oxygen deficiency; (8) the development of cancer runs contrary to all biological laws, thus being inexplicable in terms of physiological processes; (9) cancer is only a local process; (10) cancer is a systemic condition; and (11) cancer can be cured by cell destruction with chemical agents or irradiation.

Finally, I state my own contentions:

(1) Cancer is a healing attempt of the organism, brought on by chronic noxae whose constant action prevents the healing attempt from being completed.

(2) The cells in the process of wound healing and the cells at the beginning of the cancer process are identical.

(3) Cancer develops when the organism's regulatory mechanisms fail.

(4) There is no such thing as a common "carcinogenic" factor - the mechanism of action of "carcinogenic substances" being always and solely a matter of constant irritation.

(5) The genesis of cancer depends on the manner in which the cell-damaging irritation is brought to bear and the duration of its action on the cell.

(6) Irritation and deficiency factors will cause the regulatory mechanism to fail (industrial noxae, abnormal nutrition, artificial fertilizers, production of adulterated foods, accumulation of insecticides in foodstuffs. etc.).

(7) Cancer cells are dedifferentiated cells that develop during the healing process - a phenomenon common to all forms of cancer. Various researchers are today using cancer as a collective term emphasizing the variety of cancers. It is my belief that there is no variety in their genesis.

(8) The very fact that cancer does not cause pain in its initial stage shows that it is not a process contrary to nature.

(9) Every one of us is threatened with death from cancer because of our inability to adapt to present-day living conditions.

(10) According to my theory, cancer will be curable by artificially inducing cell division.

I now discuss the genesis of cancer from the viewpoint of a frustrated healing attempt of the organism.

It is a well-established fact that, with the aid of chemical or physical factors, cancer can be produced at will. TABLE 1, listing known carcinogenic factors, cites the many irritants by means of which tumors can be produced.

This list, which cannot even be considered complete, leads us to the conclusion that the term carcinogen has by no means the importance

TABLE 1

KNOWN CARCINOGENIC FACTORS

Chemical and Other Factors

Arsenic			
Asbestos			
Chromate			
Aniline derivatives		Benzidine	
		2-naphthylamine	
		0-amidoazotoluene	
		Butter yellow (*p*-dimethylaminoazobenzene)	
		2-aminofluorene	
		2-acetylaminofluorene	
		2,3-azotoluene	
		p-benzoquinone	
		4-dimethylaminostilbene, etc.	
Tars	Coal tar	Oils	Mineral oils
	Lignite tar		Shale oil
	Tobacco tar		Paraffin oil
	Wood tar		Anthracene oil
	Turpentine tar		Argillite oil
	Shale tar		
	Gas tar	Pitches	Pitch oil
	Charcoal tar		Tar gas
	Pine tar		Creosote
	Coffee tar		Naphthalene
Carcinogenic constituents of tar		10-methyl-1,2-benzanthracene	
		9,10-dimethylbenzanthracene, etc.	
		3,4-benzpyrene	
		1,2,3,4-dibenzpyrene	
		3,4,8,9-dibenzpyrene	
		3,4-benzophenanthrene	
		2-methyl-3,4-benzophenanthrene	
Other cyclic compounds		Methylcholanthrene, etc.	
		10-methyl-1,2-benzacridine, etc.	
		1,2,7,8-dibenzacridine	
		"Styryl 430"	
		Indole	
		Stilbestrol, etc.	
Synthetic materials		Plastics (implanted)	
		Rubber (implantation of pessary fragments)	

TABLE I (continued)

Simple chemical substances	Glucose solution (20-25% s. c.)
	Common salt solution (5-25% s. c.), etc.
	Urethan
Parasites	*Schistosoma haemotobium (Bilharzia)*
	Opisthorchis felineus
	Distomum japonicum (liver leach)
	Spiroptera neoplastica
	Cysticercus fasciolaris
	Nochtia nochti (nematode)
Virus agents	Rous virus
	Virus in leukemia of fowl, etc.
	Shope papilloma virus
Deficiencies, without application of "carcinogenic substances"	Choline deficiency diet
	Oxygen deficiency *(in vitro)*
	Physical Factors
Burns	
Radiation	Ultraviolet rays
	X rays
	Alpha rays
	Beta rays
	Gamma rays

ascribed to it. If we are to regard not only chemically pure compounds such as benzopyrene or butter yellow but also physiological substances such as glucose and inorganic salts, viruses, bacterial animal parasites, and other irritants as carcinogens, then there can hardly be such a thing as a *universal* specific carcinogenic factor.

Upon examination of this list, one must be convinced that the action resulting from these diverse factors must be caused by the target and not by the substances themselves. The only thing common to all these factors is their ability to produce a chronic irritation with all gradations of intensity, differing only in the speed by which they transform normal cells to malignant cells. This shows that the origin of cancer must have its inception in the chronic irritation of somatic cells. The organism reacts in the same way as in the process of wound healing, namely, by defense mechanisms seeking to re-establish the disturbed harmony; that is, by trying to heal. The cells respond by dedifferentiation and more frequent mitosis so that new tissue can be formed, but they are still controlled by the over-all regulating system of the organism

and do not continue to grow autonomously since, when the healing is completed, they revert to normal differentiated tissue cells. Growth is controlled by the organism as a whole, and the healed tissue remains "normal." This process fails to be consummated if the irritations continue to act incessantly on the cells engaged in the healing process. These cells become even more dedifferentiated with ever-increasing frequency of mitosis. After a latency period of many cellular generations, the dedifferentiated cells gradually lose their power of redifferentiation. These cells are then no longer subject to the regulatory mechanism of the organism but are now autonomous, that is to say, cancer cells. They have lost their specific functions and retain only the functions of nutrition and reproduction essential to self-preservation. These cells – now cancer cells – retain no correlation to the organism as a whole other than that of blood supply and nourishment. At this stage no further cellular irritation is required, the process then being irreversible (FIGURE 1).

I am aware of the simplification of this diagram, ignoring the complexity of the process of wound healing, which represents one of the true wonders of nature with its mysterious directive force.

When we use the expression wound healing, our thinking usually restricts the meaning of this term to the repair of mechanical lesions of the body. However, this process must also have an equivalent on the cellular level, that is, the restoring of intracellular "wounds," extending even to the molecular level. Thus, the expression wound healing comprises not only a variety of possible lesions but also a variety of regenerating mechanisms.

Let me mention the work of Glücksmann (1951) in which the experimental situation allowed an unambiguous analysis. Glücksmann irradiated mice with an electron beam in doses sufficient to cause ulceration. He found: "Under these conditions the subcutaneous tissue is unable to form a stable scar. Though proliferation of subcutaneous fibroblasts and epithelial regeneration allow an early healing of the radiation ulcer, this scar breaks down, and, though there is again scar formation from the subcutaneous tissue, this, too, proves unstable. The connective tissue formed by the subcutaneous tissue degenerates to an avascular, almost acellular and hyaline state. This tissue is partly lysed, and at this time the overlying epithelium becomes atrophic, detaches itself from the scar tissue below, and the epithelium together with the hyalinized and lysed scar tissue is shed. This process is followed by another attempt of scar formation and subsequent breakdown and leads ultimately to malignant transformation of the overlying epithelium, which occurs after about fourteen months."

I think that these results are only one example of the variety of modifications of the wound-healing process that terminates in the formation of malignant cells. I feel it desirable to emphasize the fact that

we must regard the process of carcinogenesis more as a modification of wound healing than as a phenomenon per se.

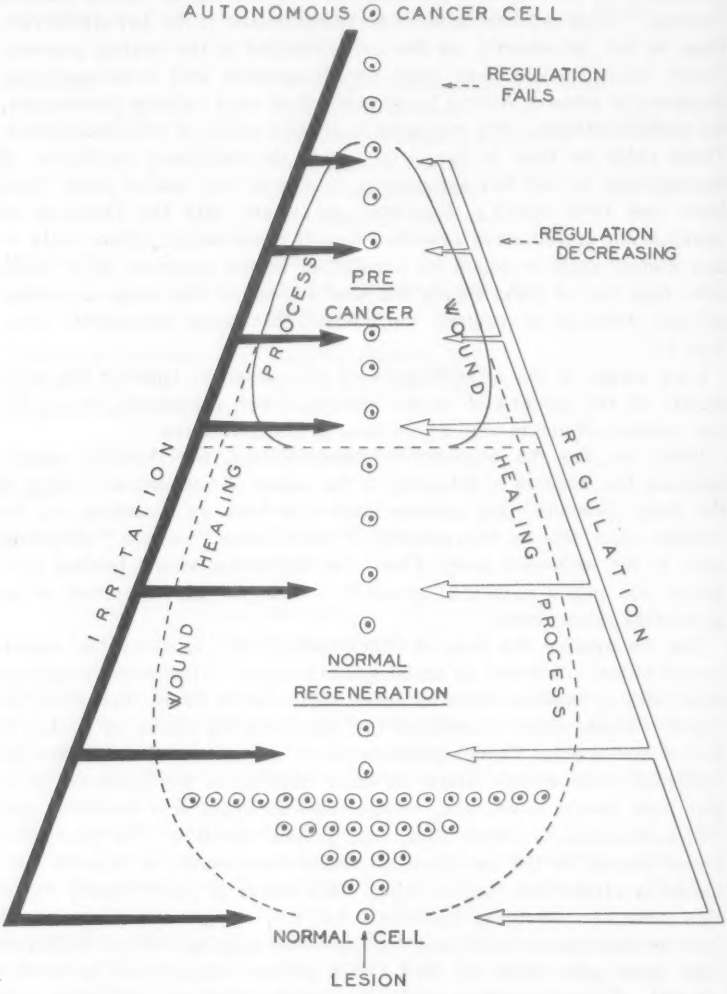

FIGURE 1. Diagram of the process of the development of cancer cells (Berglas, 1957).

The crucial events in all these processes are those of differentiation, dedifferentiation, and redifferentiation. In the process of the organism's development, the qualities inherited by the germ cell fan out to form the

differentiated cells, each cell, endowed with its specific properties, being responsible and indispensable for the harmonious interplay in the symbiosis of the multicellular organism. This differentiated organism does not represent a static system, as there is a continuous flux. The apparently constant shape of the organism is the result of an equilibrium between processes of degradation, transformation, and reconstruction of its constituents, directed always toward maintenance of life.

Dedifferentiation during wound healing is a transient event during which function is temporarily replaced by self-duplication, but the cells return to their inherited state, as predestined by the germ cell. In contrast to the redifferentiation taking place at the end of the wound-healing process, the ability to redifferentiate is lost when the healing process is frustrated by continuous irritation: the dedifferentiation proceeds and gradually becomes irreversible. Dedifferentiation, as found in malignant cells, is not the return to the potentiality of the germ cell or of embryonic cells but is, on the contrary, a loss of this ability. Deriving from a differentiated somatic cell, malignant cells may still contain traces of their origin and may still possess parts of functional activity; however, their main quality is no longer functional activity but self-reproduction. I quote Schultz (1958): "In the tumor cell, it is almost as if the chromosomes have, as their main function, their own replication."

We are aware that every frustrated process of repair involves the possibility of an aberration of the differentiation process that may lead to cancer, and the question arises whether we can avoid this threat. In order that our organism's extremely complex and delicately tuned defense mechanism may function properly, a harmonious interplay of innumerable factors is required. In this respect I mention the interplay of the autonomous nervous system, the humoral system, the endocrine glands, and cellular metabolism. This harmonious interplay, which is the result of millions of years of evolution of optimal combinations, has been interrupted, especially in recent decades, because many of the known carcinogens, with the progress of civilization, have entered the environment of man. An increasing number of noxae, such as air pollution, exhaust gases and smoke, industrial noxae, tobacco, chemical pretreatment of plants and foodstuffs, as well as radiation and nuclear fall-out, and many other factors all threaten man during his lifetime. Furthermore, the progress of medicine and hygiene has increased the life expectancy of man and thus favored the development of cancer, which demands a latency period for its realization. The last decades have brought about drastic changes in our environment, and no one can predict whether the changes in future generations may not even be more drastic. The thought that it would be possible to change our environment in such a way as to avoid all these by-products of our present-day civilization is Utopian. However, must we pay for this kind of progress at the price of suffering cancer? The question arises whether the human

organism is capable of adapting to its many harmful environmental influences. In experiments with short-lived organisms such as bacteria, we have observed the acquisition of resistance against an unsuitable environment, one example being the development of strains resistant to certain antibiotics. Similar adaptation has been observed in insects that have developed a resistance to dichlorodiphenyltrichloroethane (DDT). Whether intracellular modifications or selective processes have been involved we do not know. In the cases of both bacteria and insects, the adaptations are transmitted by heredity. From these recent observations it might be supposed that man might similarly adapt to the ravages of civilization. Since the human individual is subject to the same laws of nature as other organisms, such adaptation, theoretically, should be possible. In analyzing this possibility and its realization, we must come to a negative conclusion as the time factor proves decisive. To understand the vital importance of the time factor, we must think in terms of biological rather than astronomical time.

Approximately 30 years equal one human generation. Adaptation to the changes that have taken place in recent decades would require at least hundreds of generations, as shown by animal phylogeny and genetics. We can assume an average period of 20 to 30 minutes for a bacterial generation. Taking this into consideration, the prerequisite for the rapid appearance of numerous individuals, as well as that of adapted individuals, is given. Among bacteria passing through approximately 330 generations, representing approximately one week in astronomical time, a strain adapted to a noxa can develop in a short time. How many generations would be required for mankind to adapt itself to the innumerable noxae already present in its environment? Since only 330 human generations represent approximately 10,000 years, we realize that we would have to count by millenia to achieve adaptation, assuming that the human race survived. As prevention of cancer by adaptation seems almost impossible, all our efforts must be directed toward its cure.

The most pronounced quality of the tumor cells is their high frequency of division. We must keep in mind that this is only a symptom of their autonomous growth, but a very peculiar one. Control of cell division (Swann, 1957,1958) includes all factors, intra- or extracellular, that guarantee a normal growth, that is, the equilibrium between all parts of the organism. The most important intracellular factor counteracting the growth tendency is the function of the cell. However, within a multicellular organism there must exist additional control mechanisms of an extracellular nature; these might be hormonal or neural factors or antitemplates in the sense of the theory of Weiss and Kavanau (1957), although as yet we have no knowledge as to their chemical nature. Even if all prerequisites for cell growth are available under optimal conditions, rapid cell division does not normally occur if it is not required. In the state of regeneration, cell division occurs as long as needed, but

essentially ceases after the repair of the defect. In contrast, the malignant cell continues to grow and to divide without consideration for the requirements of the host organism. The cancer cell is dedifferentiated, autonomous, and parasitic in its nature.

Cell division is a very simple event if we look only at the end result: the formation of two new cells from one. The more we go into details, however, the more complex this basic phenomenon of the life cycle of the cell becomes, and no one can deny that this phenomenon is still one of the greatest riddles of nature. The morphologic description of this event, as found in the textbooks, gives us the impression of a complete understanding of all the steps involved in cell division. This is in error. In reality, this description tends to simplify a sequence of single events, each of which must be the result of a teamwork of biochemical units within the cell. I refer to the new investigations of Mazia (1956) and others that have brought a fresh impetus to a field where stagnation gave the illusion of established knowledge. In recent years, cytochemistry has demonstrated the role of desoxyribonucleic acid (DNA) as the prerequisite for cell division. DNA, as an important building block of the chromosomes, must have been doubled before cell division occurs. In addition, the studies of Mazia show that the synthesis of the spindle proteins must also be accomplished before cell division begins, so that a cell with doubled DNA cannot divide before the preparation of other systems essential for the mechanism of cell division is completed. All this demonstrates that our present knowledge of the biochemistry of cell division is insufficient to give a complete description of this phenomenon, even under so-called normal conditions. Even less does our knowledge suffice to describe the abnormalities we can observe under pathological conditions as, for example, in tumor cells.

In recent years great attention has been paid to the phenomenon of aneuploid cancer cells, and it has been shown that variations of chromosome number and chromosome type exist in many tumor cells. While normal cells generally divide into two, tumor cells may perform divisions into three, four, or more daughter cells. In this case the distribution of the chromosomes is not regular, resulting in cells with different numbers of chromosomes. A good example of the great variety of chromosome numbers in the individual cells, that is, the phenomenon of aneuploidy, is to be found in the work with Yoshida rat tumors (1952), showing chromosome numbers between 29 and 102 (TABLE 2).

This distribution of chromosomes has been found in a tumor line developed following the transplantation of a single tumor cell. Such a wide range of variations cannot be the result of multipolar divisions only, but would indicate that bipolar divisions also must have been connected with an irregular distribution of the chromosomes. Despite this abnormality of chromosome numbers, most of those cells are viable and able to produce tumor cell populations after transplantation. As an

TABLE 2

VARIATIONS IN NUMBER OF CHROMOSOMES IN CELLS OF YOSHIDA SARCOMA AFTER SINGLE-CELL GRAFTING*

Number of chromosomes	*Percentage of cells*	*Number of chromosomes*	*Percentage of cells*
29	1	44	5
32	2	45	9
33	3	46	10
34	4	47	4
35	2	48	3
36	6	49	5
37	3	50	3
38	5	54	1
39	2	56	1
40	4	57	1
41	4	73	1
42	11	91	1
43	7	102	1
	54%		46%

*Reproduced by permission from Lettré, 1953.

example, in the case of the Yoshida tumor, 55 percent of single cell transplantations gave takes. Among the cells that take, no specific number of chromosomes is present in such a high percentage; thus, we must conclude that even cells with a great deviation of the chromosome number are able to muitiply. Makino (1957) discussed the so-called stem cell theory, assuming that only certain types of tumor cells in a population, maintaining a constant number of chromosomes, are able to divide, and that these so-called stem cells are the real malignant cells. As yet, it has not been established whether all types of tumors are aneuploid and, therefore, whether the number of stem cells might also be variable in different tumors.

My concept of a tumor therapy is based on the following: up to now we have tried to control tumor growth through inhibition of the growth of the tumor cells or of cell division. In contrast to this, I have followed the concept of inducing such a high frequency of division on the part of the cancer cell that it will divide itself to "death," becoming unviable or losing its drive to divide rapidly, and thus becoming harmless. At first, this idea seems to be a paradox; therefore, let us now examine it. A tendency to divide is one of the most characteristic features of malignant cells, and it is here that the most specific difference from normal cells can be seen. It is specifically this tendency that should be taken advantage of in considering a therapy. The growth of a cell, normal

or malignant, occurs by doubling of all essential components before cell division. Only then can a division produce two new complete cells. For successful therapy, stimulation of cell division should not mean stimulation of the entire system, as this would lead to an increase of viable tumor cells. Stimulation of cell division should mean that mitosis is induced at will, at a time at which the synthesis of all cell components has as yet not been completed. This kind of cell division can be called premature cell division. The result of premature cell division cannot be foreseen in all its consequences. In any event, we can expect the formation of incomplete cells; whether the resulting daughter cells are still viable depends on the degree of incompleteness.

Our problem is whether it is possible to induce premature cell division at will. According to our present knowledge, the cell divides only after completing all necessary prerequisites of growth. It is still an unsolved problem whether cell division occurs inevitably after the completion of all prerequisites or whether an independent "trigger" is necessary to start cell division. As long as this basic question is open, we cannot know whether a trigger might induce cell division at any time, independent of the stage of synthesis inside the cell. My proposal to provoke premature cell division raises many important cytological questions.

Many years ago it was shown by Hartmann (1928) that, if cytoplasm is repeatedly amputated from a growing cell, so that it cannot complete its growth, it will remain alive, but will not divide. Thus, artificially a situation is created that can be maintained indefinitely. This result can be interpreted to mean that, when the cell's content of all cytoplasmic constituents is reduced periodically, the trigger system, if this exists, is also reduced. In this respect the experiments of James (1953) are enlightening. James found, in *Amoeba proteus*, that if he amputated as much as one half of the cytoplasm of a cell at a time when it was about to enter prophase, he could no longer stop the division. The reduced cell divided to give two small daughter cells. Obviously, the trigger had already worked; the decision to divide had been made so irrevocably that even reduction in cytoplasmic mass could no longer stop division.

Whether reduction of nuclear mass under proper conditions would allow cell division is unknown, but this possibility cannot be excluded. The mechanism of division should be able to work at any time without the result of producing equal and complete cells. A more intense study of the phenomena of cell division, of the mitotic apparatus, and of the role of the centrioles (Lettré and Lettré, 1958, 1959) is necessary. It must be of value to study the induction of mitosis in polyploid cells and of the reduction-division in meiosis.

Thus far we have studied cell division as a physiological phenomenon with the goal of maintaining life. In tumors we observe abnormalities of cell division that are not found in the physiological range, but the

growth of the tumors is not inhibited. Premature cell division would be a variation of the process of growth, without consideration for the maintenance of the cells, and would therefore mean their own destruction.

My hypothesis of the development of malignant cells by chronic irritation and altered cell division leads to the proposition of introducing premature cell division for the therapy of cancer. It remains to be seen whether it will be possible to find an agent that induces premature cell division only in tumor cells. Although this would be desirable, it does not seem indispensable because, as tumor cells have a higher rate of division than normal cells, they should be more sensitive to stimulation even with a nonspecific agent. It is logical to assume that, even if normal cells are also stimulated, the malignant cells will be the first to perish. In this respect, it is not of great importance whether the stem cell theory is correct; if it were, the therapy would not be required to destroy all tumor cells, but only the stem cells.

It has been the purpose of this presentation to expound, first, my theory of the genesis of cancer and, second, my proposal for a therapy based on an artificial increase in the rate of mitosis of cancer cells. In spite of the importance of all other fields, it seems reasonable that basic research in the field of cell biology should have priority in research programs. The importance of the research worker in every area of science is evident, and all questions raised in regard to the cancer problem will find their solution only through experimental work. International cooperation on a world-wide scale in all the different disciplines is essential in order to achieve our goal: the cure of cancer.

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DIVISION OF NUCLEAR PHYSICS

THE WORLD-WIDE IGY COSMIC RAY PROGRAM*

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The Cosmic Ray Program of the International Geophysical Year was organized to coincide with the period of maximum solar activity expected to occur during late 1957. While cosmic rays are not produced in substantial numbers by the sun, the intensity of the radiation is greatly affected by solar activity.

The sun is known to have a 11-year cycle of "activity." By activity we mean the number of spots and flares observed on the solar disc. As expected, a maximum did occur during September 1957, and the activity remained high during all of 1958. This maximum appears to have been the most active ever recorded. Solar activity records extend back for over 300 years.

Because a broad maximum was anticipated, an 18-month "year" was agreed upon. Thus, the IGY lasted from July 1, 1957 through December 31, 1958. It has been followed by another program called International Geophysical Cooperation 1959, or IGC-'59. Most of the nations in the world have cooperated in these programs.

An international table of organization is shown in FIGURE 1. We see that the United States National Committee has a total of 17 technical panels reporting to it, of which the Cosmic Ray Technical Panel is one. This panel is composed of investigators competent in the field of cosmic ray research, and is responsible for the detailed development of a research program in this field for the United States.

The major task of cosmic ray research during the IGY was the measurement of the intensity during periods of solar activity. In order to do this, two standard instruments were designed and agreed upon for international use at sea level and mountain altitudes. These are (1) the neutron monitor and (2) the meson telescope. These instruments record the data every 15 min. except during a solar flare, when data are recorded more frequently. The records, after suitable corrections for atmospheric pressure and temperature fluctuations, are sent to the various World Data Centers. Center A is located in the United States and Center A (cosmic rays), for example, is at the University of Minnesota, Minneapolis, Minn. Scientists over the world are then able to analyze the data taken by identical instruments at the same time all over the globe.

*This paper, illustrated with slides, was presented at a meeting of the Division on November 11, 1959. The research described in this article was supported in part by the joint program of the Office of Naval Research and the Atomic Energy Commission, and in part by the United States National Committee for the International Geophysical Year.

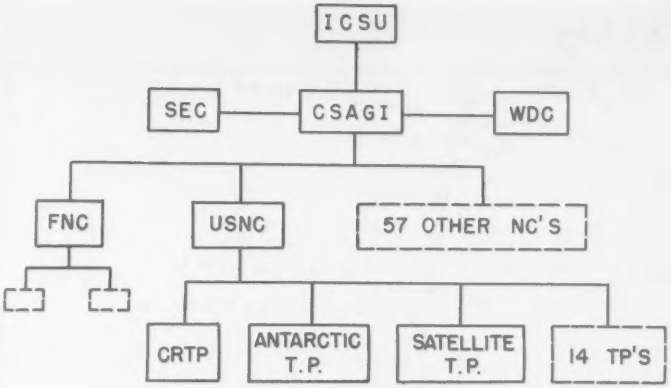

FIGURE 1.

One of the major problems facing the CRTP was the geographical location of this network of stations. FIGURES 2 to 4 show the stations upon which agreement was finally reached. At New York University, for example, we operate a neutron monitor at College, Alaska, about 6 miles northwest of Fairbanks. This location is of particular interest because it is in the zone of maximum auroral occurrence. A similar zone is found surrounding the south geomagnetic pole.

Besides this ground-level work, considerable effort has been expended to expand our knowledge of cosmic rays at high altitudes. This research has been conducted with the aid of balloons, rockets, and satellites.

Modern research balloons are built of polyethylene 0.001 in. thick (or less), and have volumes of as much as 8 million cubic feet. Such a balloon is 380 feet long, and is capable of lifting a 200-lb. payload to approximately 140,000 feet. It is capable of floating at this altitude, where 99.8 per cent of the earth's atmosphere is below the balloon, for many days. This long exposure time makes it a very useful vehicle. Also, it is quite economical to make and operate in comparison to a rocket or satellite.

A sounding rocket of the Astrobee type, for example, can lift a 40-lb. payload to an altitude of 800 miles. While this additional altitude is most desirable, the major shortcoming is the brief duration of the flight, which is only about 10 min.

Satellites are very useful vehicles for high-altitude cosmic ray research. They can expose the apparatus to the primary radiation for very long periods of time if the orbit is such that atmospheric drag is slight. With such vehicles, it is possible to examine the direct effects of a solar eruption. Thus far, the major disadvantage of the satellite has been the limited payload capacity. The first such satellites were launched during 1957 as part of the IGY program.

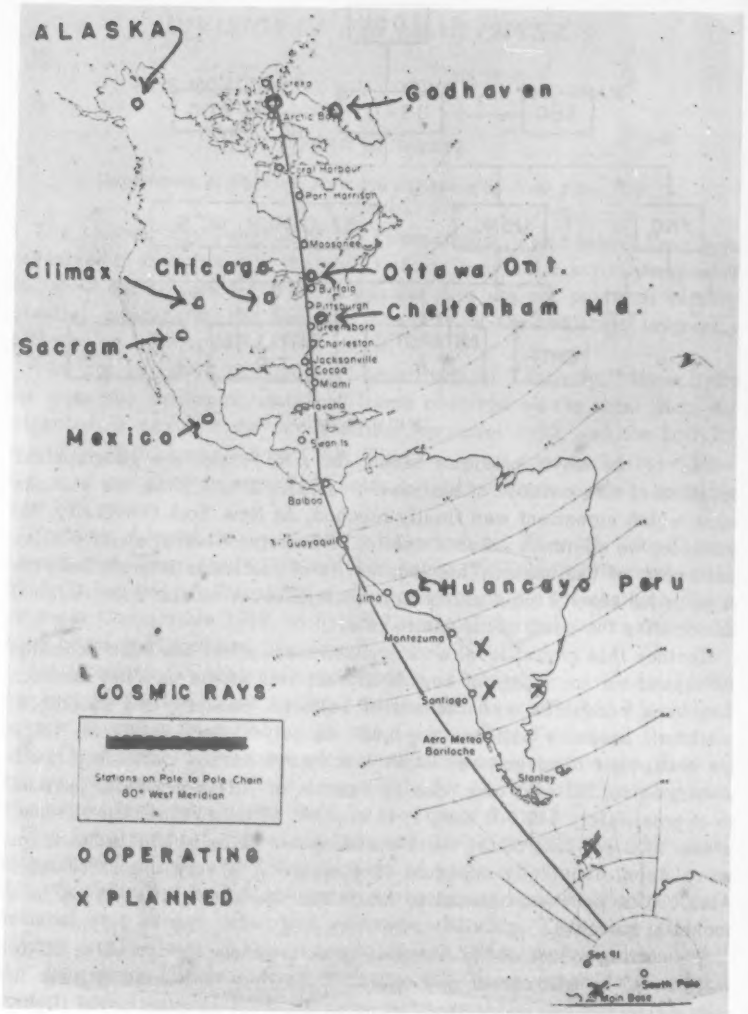

FIGURE 2.

The results of this research have been impressive, and analysis of the data has only begun. Among these results we may list the following:

The Van Allen radiation. J. A. Van Allen of the University of Iowa, Iowa City, Iowa, was the first investigator to detect the existence of this radiation at satellite altitudes. This radiation, presumably composed of protons and electrons, is found in belts surrounding the earth.

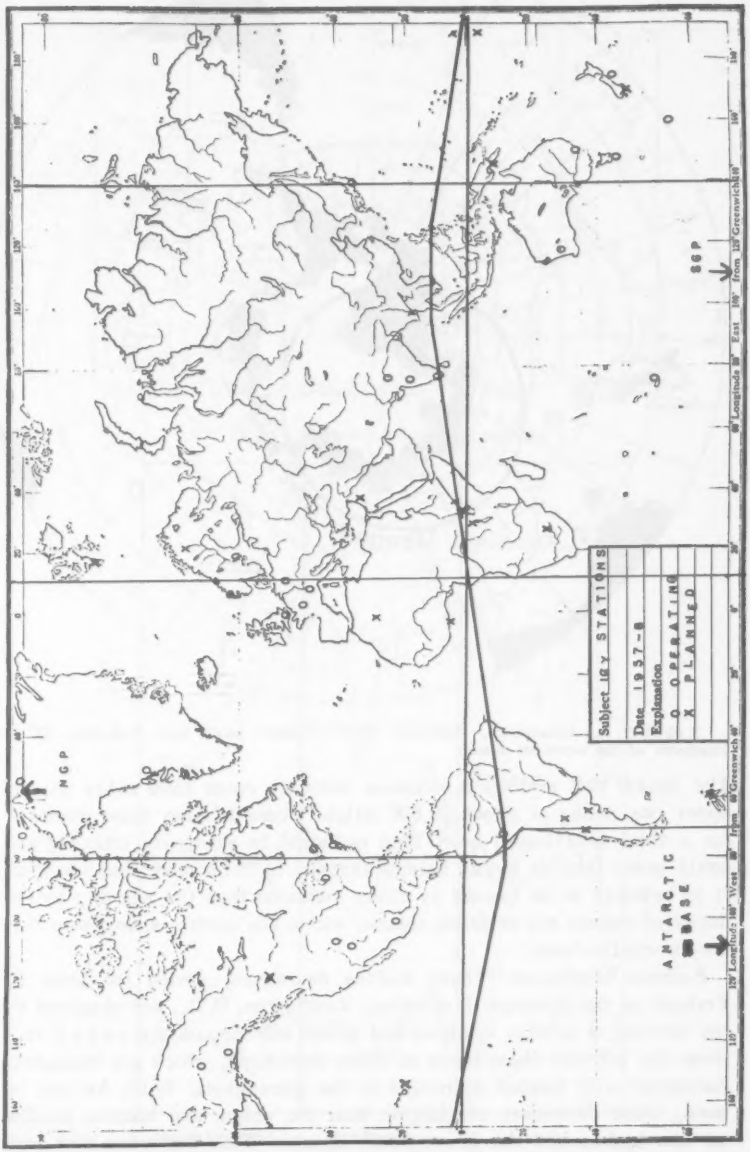
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FIGURE 3.

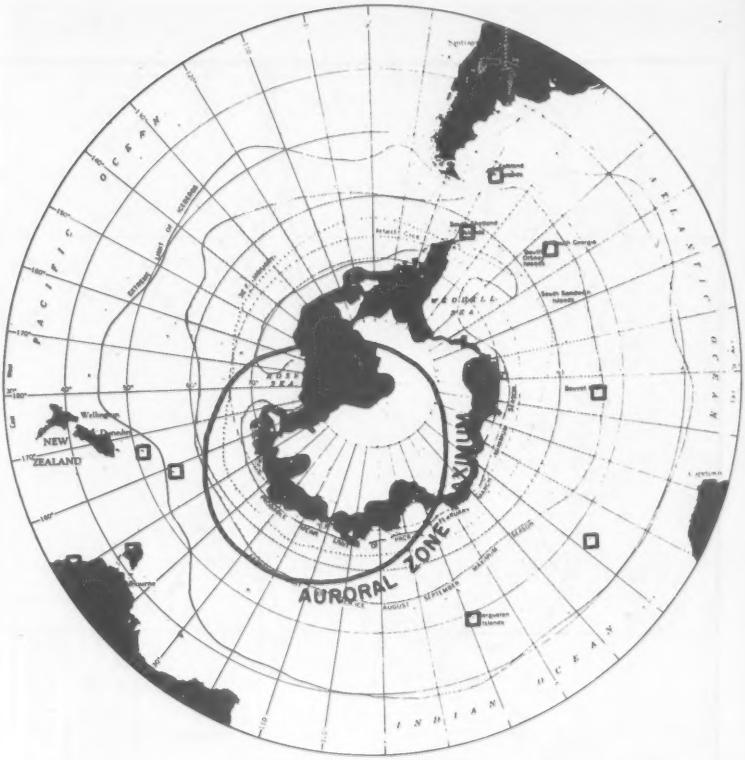

FIGURE 4. Antarctica. Relative accessibility: pack ice, icebergs, 32° F. isotherm of the warmest month.

The lowest belt reaches a maximum intensity about 1500 miles up; the upper one peaks at about 20,000 miles. There is also some evidence for a third (and lower) belt. This radiation is extremely intense, and could prove fatal to human beings exposed to it for more than an hour. It is believed to be caused by direct emission from the sun and by the decay of cosmic ray neutrons created within the earth's atmosphere that are directed outward.

Forbush decreases. These sudden decreases, named for Scott E. Forbush of the Carnegie Institution, Washington, D.C., are observed by the network of neutron monitors and meson telescopes. FIGURES 5 to 7 show the latitude dependence of these decreases, which are intimately connected with sudden decreases in the geomagnetic field. As may be seen, these decreases are largest near the poles, and become smaller as one approaches the geomagnetic equator. This indicates that low-energy particles are predominantly affected.

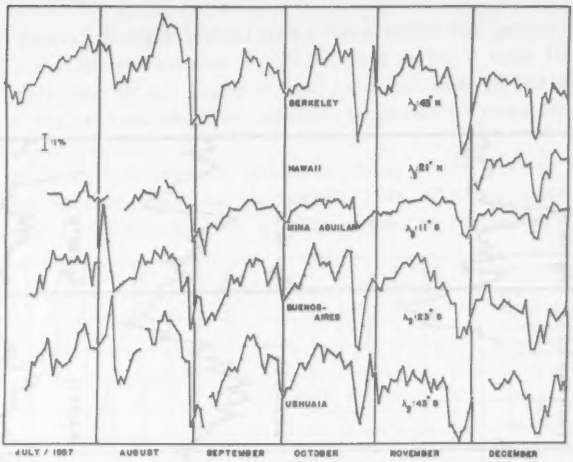

FIGURE 5. IGY neutron monitor records.

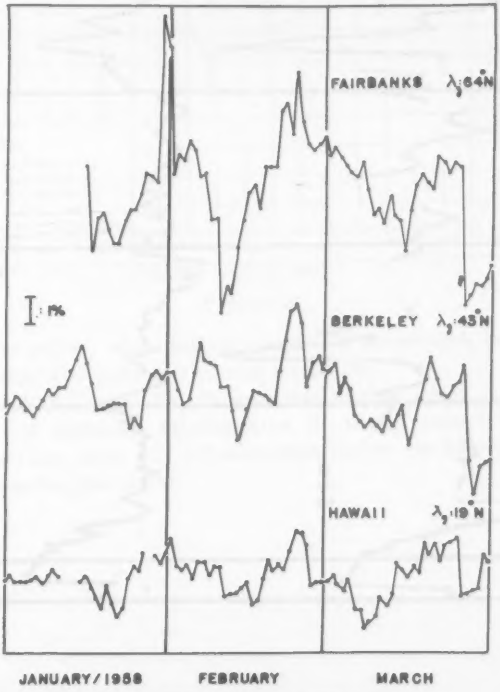

FIGURE 6. IGY neutron monitor records.

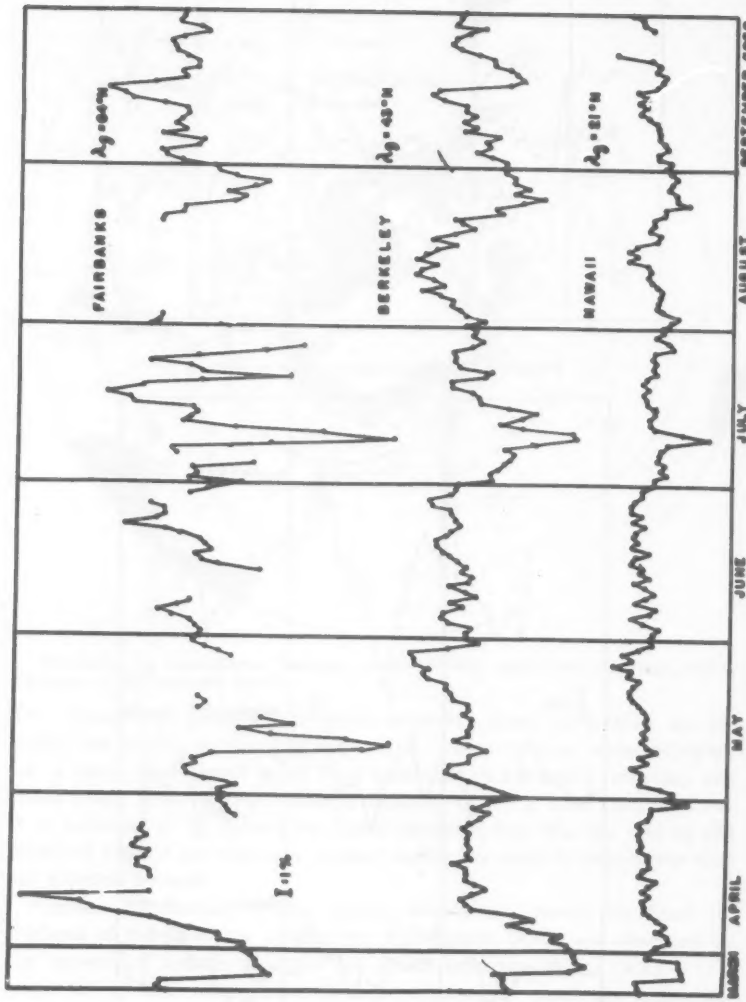

FIGURE 7. IGY neutron monitor records.

X-ray bursts. Several investigators have noted the presence of intense X radiation at altitudes of 100,000 feet during a solar flare. This intense radiation is not found at sea level because of the absorbing effects of the atmosphere. This radiation is probably caused by electron emission from the sun.

Our own work has shown a sudden doubling of the neutron intensity at balloon altitudes just before sunset. This effect is illustrated in FIGURE 8. The intensity then quickly returns to normal as sunset is

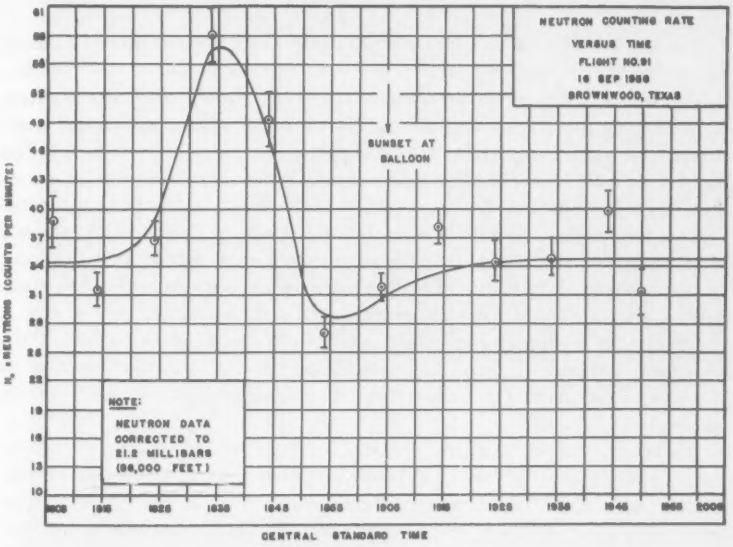

FIGURE 8.

reached. The effect, while not yet completely understood, is most probably connected with particle emission by the sun.

Thus, the IGY Cosmic Ray Program has already added greatly to our knowledge of earth-sun relationships. It seems certain that further analysis of these data will enhance even further our knowledge of the space surrounding our planet.

DIVISION OF INSTRUMENTATION

IN VIVO DOSIMETRY WITH MINIATURE RADIOLUMINESCENT GLASS RODS*

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Veterans Administration Hospital, Bronx, N.Y.

The need for an *in vivo* dosimetric system has been evident to radiologists for some time. Almost three years ago we surveyed the existing dosimetric systems in order to select one that would meet the requirements of any good dosimeter and yet would be of minimum size and comprised of nontoxic materials. Minimum cost was also to be considered. The system selected is based on the principle of radiophotoluminescence[1] developed by James Schulman and his colleagues.[2] The dosimeter selected is a glass needle 6 mm. high and 1 mm. in diameter, comprised of a glass host matrix consisting of either barium-potassium, aluminum metaphosphate, or lithium, aluminum manganese metaphosphate. Regardless of matrix composition, silver metaphosphate is added.

Absorption of energy can result in three effects: (1) dissipation of the absorbed energy as heat; (2) reradiation of the absorbed energy as luminescence; and (3) production of photochemical changes in the absorber.

Whenever a solid, such as a glass, is exposed to ionizing radiation, varied effects take place. The following phenomena must be considered: (1) the radiation field creates ionization electrons in the glass; (2) for each ionization electron, one electron-deficient region or hole is formed; (3) adsorption bands due to centers are formed by electron trapping; and (4) competition for ionization electrons exists between holes and the various kinds of electron traps.

When glass is subjected to ionizing radiation, the principal effects will result from electronic processes. These effects occur because electrons are excited sufficiently to leave their normal positions and move through the glass network. In some glasses, these ionization processes will be purely transient. In other glasses, the electron distribution will be permanently altered.

* This paper, illustrated with slides, was presented at a meeting of the Division on November 17, 1959.

The Section of Chemical Sciences held a meeting on November 3, 1959 at which Raymond M. Fuoss of Yale University, New Haven, Conn., presented a paper entitled "The Conductance of Simple Electrolytes." This paper will not be published by the Academy.

The Division of Psychology held a meeting on November 16, 1959 at which Nathan S. Kline of Rockland State Hospital, Orangeburg, N.Y., presented a paper entitled "Psychiatry and Psychiatric Organization in the Soviet Union." This paper will be published in the *Annals* of the Academy.

The ionization process may be described as follows: photons interact with matter in three ways, depending upon their energy, and produce photoelectrons, Compton electrons, or positronelectron pairs. In this way a large fraction of the energy of the photons or gamma rays is transferred to electrons and positrons. Since so few positrons are produced by most gamma-ray emitters, they will not be explicitly mentioned further. In passing through the glass network, energetic electrons lose energy by interacting with the less tightly bound electrons, mostly by means of Coulomb forces. If this interaction is strong enough, bound electrons are ejected from their normal orbits with sufficient energy to cause them to move through the network.

Electron-deficient regions, or holes as they are usually called, most probably move through the network until they are trapped. In crystals, and most probably in glass networks, hole trapping occurs very quickly, perhaps in one tenth or one one hundredth of the time required to trap electrons. In addition to being trapped, the holes may migrate through the network until they encounter electrons with which they recombine, thus effectively destroying both a hole and an electron. If holes are trapped as quickly as is believed, the latter process results in only a small fraction of the recombination that occurs. In a similar manner, electrons move through the network until they are trapped or recombine with holes. Electrons may also be trapped by impurities in regions of net positive charge. The recombination of mobile electrons with stationary holes is probably one of the dominant processes that result in the return of ionization electrons to their normal lattice positions. Each of the processes described occurs with its own probability.

In crystals, the minimum energy required to remove an atom from its normal lattice position is approximately 25 ev. This amount of energy can be imparted to the atoms in a glass being bombarded with gamma rays or X rays by the electron recoils, a process that will not make defects efficiently. Recoiling electrons rapidly lose energy and are quickly degraded below the minimum energy required to produce a displacement. Even if the electron has sufficient energy, it may make a "glancing" collision that will not transfer the necessary energy to the lattice atom.

Since there is more than one electronic process and, in particular, because several different centers may trap electrons, the capture of each ionization electron is determined by a competition between centers. To understand how this competition operates, consider that the mean-free path for an ionization electron is inversely proportional to the product of the number of centers and the capture cross section of each center when velocity and scattering effects are neglected. If only one type of center is operative, the mean-free path for electrons is proportional to the reciprocal of the product. When more than one center is present, the mean-free path is proportional to summation of this inverse product

extended over all centers. In this case, it is assumed that once a center has trapped an electron, it does not enter into further competition for electrons. Thus if some of the centers have trapped electrons, only the remaining empty centers need be considered.

Recombination of the various ions, be they electrons or holes, is possible. This recombination may be direct or indirect. Indirect trapping takes place when newly formed holes recombine with previously trapped electrons.

The principal processes involving ionization electrons may be summarized as: (1) electron trapping; (2) direct recombination; and (3) indirect recombination.

Przibram recognized that the alteration of luminescence produced by ionizing radiation consisted, in effect, of the actual creation of new luminescent centers by the photochemical action of radiation. He therefore defined the term radiophotoluminescence to describe the phenomenon where a material, originally nonluminescent under ultraviolet or visible light, responds to such excitation by exposure to ionizing radiation. To complete this definition, the following restriction is necessary: radiophotoluminescence produces new stable luminescent centers that are not destroyed by the long wave-length radiation necessary to produce luminescence.

The deliberate addition of impurities to certain solids, crystals, or glasses permits the formation, postexposure, of new types of centers involving the impurities in addition to the centers formed from the constituents and defects of the host matrix. As a result, centers formed upon exposure of the host may be either unstable or nonluminescent; the centers formed from the impurity contained in the same host matrix may be both stable and luminescent. The net effect is radiophotoluminescent.

The excitation and emission bands observed in the crystal not exposed to ionizing radiation are due to positive silver ions, while those in the exposed crystal are due to reduced silver or atomic silver of valence zero. These atomic silver centers are formed when the positive silver ions trap electrons that have been "liberated" by the absorption of the ionizing radiation. Electrons trapped by the positive silver ions are more strongly bound than those trapped at halogen ion vacancies, and hence are far more stable than the F centers. The effect of light absorption in these centers is to raise the electron to an excited state from which it returns with luminescent emission. The electron is not freed, with consequent destruction of the center. This phenomenon occurs not not only in NaCl, but in other alkali halides, potassium sulfate, and in glasses containing silver as an impurity and in NaCl:Cd.

The properties just described can and do serve as the basis for a dosimetric system. Practical consideration dictated the use of a glass. The one chosen initially is comprised of silver-activated potassium,

barium aluminum phosphate glass in which the ionizing radiation-induced photochemical reaction $Ag^{+}+e^{-}$——Ag° produces the stable atomic silver centers.

The behavior of the microdosimeter may now be described very briefly. The glass is exposed to X or gamma rays that produce ionization and a number of electrons. There is competition for these electrons, resulting in the net effect of interest, the formation of atomic silver, stable luminescent centers. This effect is directly proportional to the radiation absorbed by the glass. Hence a linear system results, capable of easy calibration results. The dosimeter is read by exciting with ultraviolet light the silver luminescent centers and converting the emitted orange-yellow fluorescence to an electric current through the use of a photomultiplier.

Biologically, the glass, even with metal shielding, is sufficiently small to be implanted in a tumorous area, using techniques developed for radium implants. Since it is comprised of glass, there will be no pharmalogical problems. Its shield must be comprised, of course, of inert metals, such as expensive Au or Pt. Note FIGURE 1.

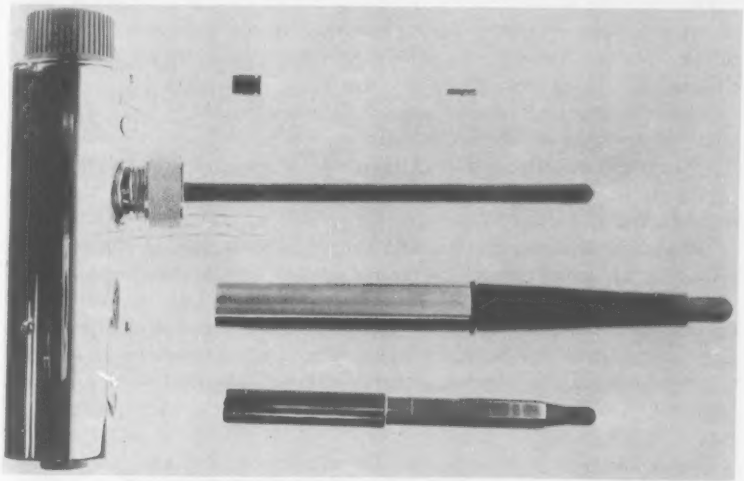

FIGURE 1. A comparison of standard air chamber, a scintillation probe (with light pipe), and the gold-shielded radiophotoluminescent needle *(upper left)*.

The glass needles are a relative system, that is, they must be calibrated against standard instruments or in known radiation fields. The use of a high Z-material shield may perturb the radiation field under measurement. However, this effect is not too great and is an acceptable trade-off in order to obtain an *in vivo* system.

Use of the needles is simple. They are read pre- and postexposure by inserting them in a fluorimeter known as a reader. One concept of

the heart of such a device developed by the Naval Research Laboratory, Washington, D.C.,[3] is employed in our reader system.

Reading prior to exposure is necessary, since thermal reduction during manufacture may produce a small number of atomic silver centers. If the radiation exposure is low, readings due to these centers could result, introducing serious errors. Such readings are termed predose and average 6 to 15 μamp. The predose is subtracted from the post-exposure reading to obtain the net reading.

Current outputs range from 5 to 2000 μamp., depending on the exposure history of the needle. Readings are obtained, using a sensitive D.C. microammeter.

Before passing on to our results, several additional points concerning these needles should be mentioned: neutron response; their use as spectrometers in a system based on adsorption spectrometry; and scanners.

Kondo, while a Fulbright fellow at Oak Ridge, Tenn., experimentally investigated the neutron response of these glasses. He found them to be relatively neutron-insensitive. We have been able to confirm Kondo's findings.

There is one striking conclusion to be drawn: that these needles may permit the measurement of gamma radiation in the presence of intense neutron fluxes. Neutron to gamma flux ratios could be 1:1 or greater. Experiments along these lines are now being undertaken at the BMRR. Exposure to neutrons and subsequent exposure to X or γ rays does not alter the response of the needles.

A complete definition of an X-ray or γ field cannot exist unless the energy distribution is known. Varying shield thicknesses are shown in FIGURE 2.

Knowledge of these factors will permit determination of the quantum efficiency of many effects, and the amounts of energy absorbed by bone, wet tissue, and fat. It is possible to determine an X-ray or γ spectrum by placing a sequence of filters about dosimeters located in a radiation field and obtaining the filtration curve. This method is known as adsorption spectrometry, a technique that has been described in British and United States literature about seven times during the last quarter century.[4-10]

The properties of X or γ rays are a function of the photon energy. Photon energy and wave length are related through Planck's constant, frequency wave length, and velocity.

The mass absorption coefficient, $\mu(E)$, may be expressed as either a function of photon energy or wave length. In the following discussion, wave length is used in order to conform with the presentation in the literature.

Dose is the time integral of intensity and in the case of a nonmonoenergetic radiation beam or field it is also the energy integral over energies present.

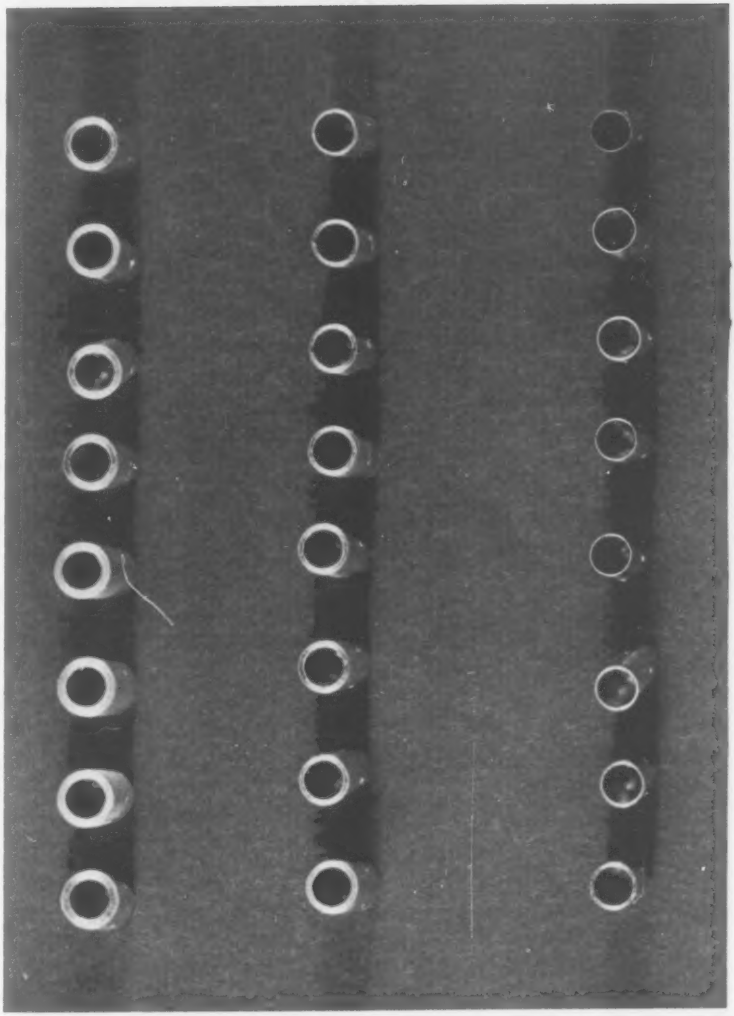

FIGURE 2. A series of aluminum filters for use in absorption spectrometry. Wall thicknesses vary from 1 mil *(lower right-hand corner)* to 24 mils *(upper left)*.

If only energy is considered, then

$$I(x) = \int_0^\infty f(\lambda)d\lambda \tag{1}$$

This is valid only for an unfiltered beam. A normalization is made so that

$$I(0) = \int_0^\infty f(\lambda)d\lambda = 1 \tag{2}$$

where $f(\lambda)d\lambda$ is the energy contained in the wave-length interval λ to $\lambda + d\lambda$. In good geometry, filter attenuation is given by

$$e^{-\mu(\lambda)x} \tag{3}$$

X is filter thickness. EQUATIONS 1 and 3 are combined to obtain

$$I(x) = \int_0^\infty f(\lambda)e^{-\mu(\lambda)}d\lambda \tag{4}$$

This is an integral equation for determining the energy distribution function $f(\lambda)$. In actual practice, $\mu(\lambda)$ or (E) is graphically known, $I(x)$ is an experimentally determined quantity, and X is the known, pre-selected filter thickness. If a curve or family of curves exists for $I(x)$ for which the integral equation can be solved, then $I(x)$ can be fitted analytically with the experimental data giving a good approximation for $f(\lambda)$. This technique is described in the literature.[3-6]

The transformation

$$f(\lambda)d\lambda = \phi(\mu)d\mu \tag{5}$$

is introduced and the integral equation becomes

$$I(x)\int_{\mu_0}^{\mu} \phi(\mu)e^{-\mu x}d\mu \tag{6}$$

The experimenter chooses the limits of integration to fit the situation under investigation.

Let $$z = \mu - \mu_0 \tag{7}$$

and $$\phi(z) = \phi(\mu) \tag{8}$$

$$\therefore I(x) = \int_{\mu_0}^{\mu} \phi(z)e^{-zx-\mu_0 x}dz \tag{9}$$

It should be noted that $\mu_0 x$ is a constant. The integral equation now takes the form

$$I(x)e^{\mu_0 x} = J(x) = \int_{\mu_0}^{\mu} \phi(z)e^{-zx}dz \tag{10}$$

Here $J(x)$ is the Laplace transform of $\phi(z)$.

An analytic function, $\bar{J}(x)$ representing the experimentally determined $J(x)$ is introduced. One form for $\bar{J}(x)$ is

$$\bar{J}(x) = a_1 e^{-b_1\sqrt{x}} + a_2 e^{-b_2\sqrt{x}} \tag{11}$$

The constants a and b are determined to match the experimental data. An approximate representation is given by

$$\bar{J}(x) = \Sigma a_n e^{-b_n\sqrt{x}} \text{ for } b_n > 0 \tag{12}$$

The spectral energy distribution can be shown to be

$$\phi(\mu) = \sum \frac{a_n b_n}{2\sqrt{\pi}(\mu-\mu_0)^{3/2}} e^{-\frac{b_n^2}{4(\mu-\mu_0)}} \tag{13}$$

The solution for this is best handled by numerical techniques and computers. Numerical methods for this have also been presented in the literature. This is not an efficient method and should be used only when scintillation techniques fail or are inapplicable.

Radiophotoluminescent glass can be used in geometrics other than that of a needle. For instance, foot-long rods could be exposed and scanned rapidly to determine the relative radiation intensity along the length of the rod. Plates have been used in a similar fashion.

The main points[10] to be considered when selecting an *in vivo* dosimetric system are:

(1) The capacity of the dosimeter to be employed under a wide range of therapy conditions (this, naturally, will require a small and complete unit devoid of connecting cables or other limiting attachments).

(2) The dosimeter should respond to a wide gamma energy spectrum with accuracy and be energy-independent, for example, from 0.250 to 1.3 Mev.

(3) The detector employed should be an integrating-type instrument so that successive daily radiation therapy doses, as well as the total radiation delivered, can be recorded.[11,12]

A partial list of the more important investigations relating to the glass dosimeter is given below:

(1) To record the daily and total dose of radiation: glass dosimeter is not discharged as a result of each reading. Daily dose and total dose can be obtained. The dosimeter is an integrating type and can be used throughout the entire course of therapy.

(2) Linearity and accuracy: *(a)* Bare glass saturates at 1200 r. with 120 kev X rays. With higher kevs, saturation will occur at higher "R" values. For Co-60 (1.3 Mev) saturation begins at 7000 r. Separate calibration curves are required. *(b)* In the past a separate calibration curve was used for a specific dosimeter and photon energy. With the shield it is possible to calibrate with any γ- or X-ray source from 0.250 to 1.33

Mev and then use the dosimeter with any source having photon energies in this range. This eliminates the requirement of calibrating a dosimeter against each specific therapy source. Saturation now begins at 10,000 r. These gold shields are easy to fabricate.

(3) Energy dependence: as can be noted from (2) above, the bare-glass dosimeter is energy-dependent. With suitable shielding material, this has been corrected for the specific photon energies listed.

(4) Orientation of glass: *(a)* in air: variation of readings by 40 per cent when long axis of the glass is parallel to radiation beam; and *(b)* in a scattering medium: less than 3 per cent regardless of orientation of glass. This is ideal, since all work will be in phantom material, then in humans.

(5) Reading time: readings: postexposure, must wait 2 hours for 90 per cent of final reading, 4 hours for 95 per cent of final reading, 24 hours for approximately 100 per cent or "true" reading.

(6) Miscellaneous information: *(a)* since the bare-glass rods will acquire an erroneous fluorescence reading due to oil from the hands, they require care in handling; *(b)* body fluids will etch the bare glass; *(c)* if more than one bare glass is used, identification is difficult unless a numbered shield is used; *(d)* there are no adverse pharmacological effects between the glass, the gold shield, and the patient; and *(e)* bleaching of glass by daylight or ultraviolet light during the normal handling and reading time will not destroy the reading of the glass rod dosimeter.

Prior to actual implants, phantoms were employed for calibration of the glass rods. Several of these types are listed below in TABLE 1.

FIGURE 3 indicates the final form of our dosimeter shield. One end is permanently sealed and an identifying number inscribed. The other end is screwed on. Four No. 80 holes are drilled 90° apart about the circumference midway from either end. The purpose of the holes is to permit entry of a certain amount of softer secondary radiation produced by the multiple scattering in the body by the primary beam.

TABLE 1

Phantom material	*Comment*
(1) Water	Not suitable, glass rods difficult to mount, erroneous fluorescence may result from glue used to mount glass rods.
(2) Wax	Not suitable. Contamination of glass with wax and erroneous fluorescence readings.
(3) Presdwood	(a) Unit density (for lung d = 0.3 g/cm.^{3}) (b) Varying density (c) Varying density (for bone, d = 1.85 g/cm.^{3}) The Presdwood phantom was selected as being the easiest to work with and maintain reproducible positions.

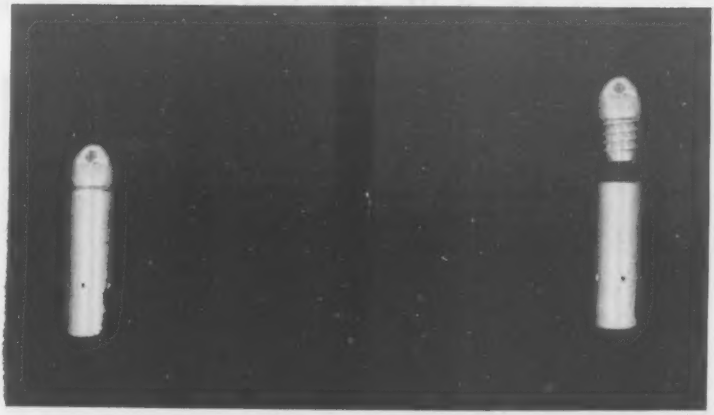

FIGURE 3. Prototype of radiophotoluminescent glass needle shield constructed of 2S aluminum having a screw-on cap and needle eye. Wall thickness was arbitrarily set at 18 mils.

FIGURE 4 presents the use of these dosimeters with hollow radium needles. The use of 2 or more of these glass rods in this needle allows for verification of the dose of radiation during a radium implant.

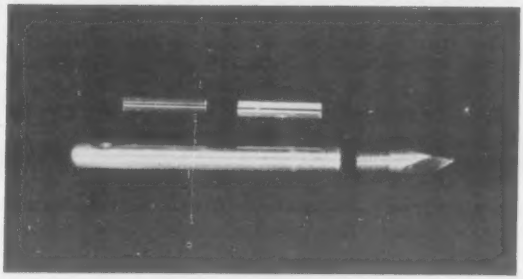

FIGURE 4. Two unshielded glass needles *(top)* and a standard, hollow, gold radium implant needle. The dosimeters are inserted into the hollow needle for implant readings.

The calibration was done, using the Ferrous-Ferric dosimeter as a standard. This procedure allowed for the measurement of absorbed dose.* This is illustrated in FIGURE 5. These glass rods were used in several radium implants, the rods being placed in hollow gold needles comparable to radium needles and implanted at specific points within the selected implant geometry. Present methods of dosage calculation

*Absorbed dose is the energy imparted to matter by ionizing particles per unit mass of irradiated material at the place of interest.[13]

such as the tube-shift method[14] and those based on analytic geometry are obviated by the use of these small dosimeters.

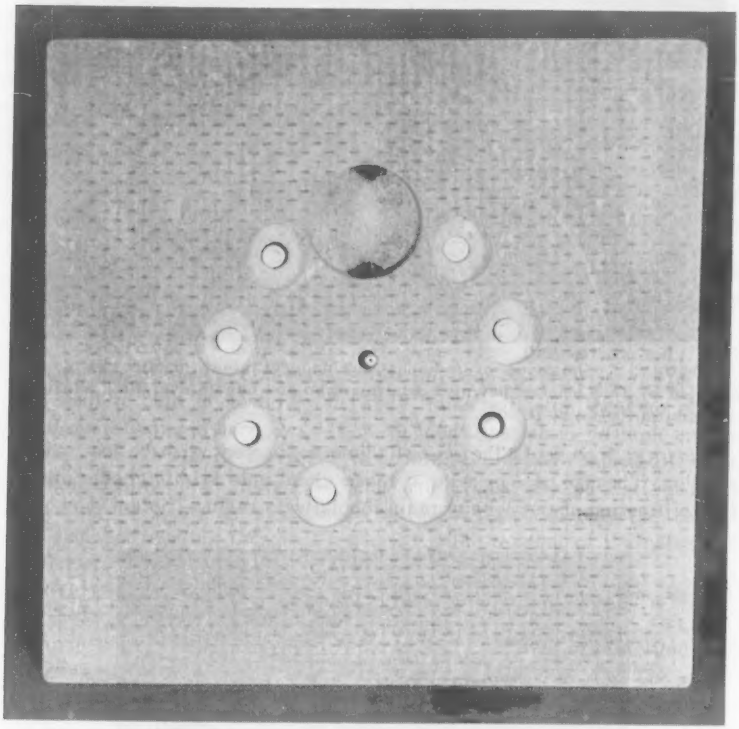

FIGURE 5. Presdwood phantom for exposing eight needles to radium or other isotope sources of radiation such as Na^{24}, Ir^{192}, and Au^{198}. Provision is made for inserting larger standard dosimeters such as a Victoreen Condensor-r-meter or chemical (Ferrous-Ferric) dosimeter.

FIGURES 6 and 7 show two cases in which the glass rod dosimeters have been employed.

Summary

The microdosimeter has an important role in human radiation dosimetry. The selection of an optimum wall thickness of the dosimeter shield permits its use *in vivo* when the photon source has an energy lying between 0.25 and 1.33 Mev independent of separate calibration tables.

The investigation of a low-Z microdosimeter for possible reduction of the energy dependance of the present glass is under study, as are the following:

FIGURE 6. Patient with cancer of bladder and 2 glass rod dosimeters inserted via urethral catheter for accurate recording of cobalt-60 radiation.

The use of the glass dosimeter with various shield thicknesses as gamma-ray absorption spectrometers.

The use of the glass dosimeters with beta-ray emitting isotopes.

The use of the glass rods to measure gamma radiation in the presence of intense neutron beams.

Use of long 25-cm. rods for scanning to determine relative radiation disturbance.

The investigation of other systems, such as thermoluminescence or semiconductors, as *in vivo* dosimeters for specific therapy functions.

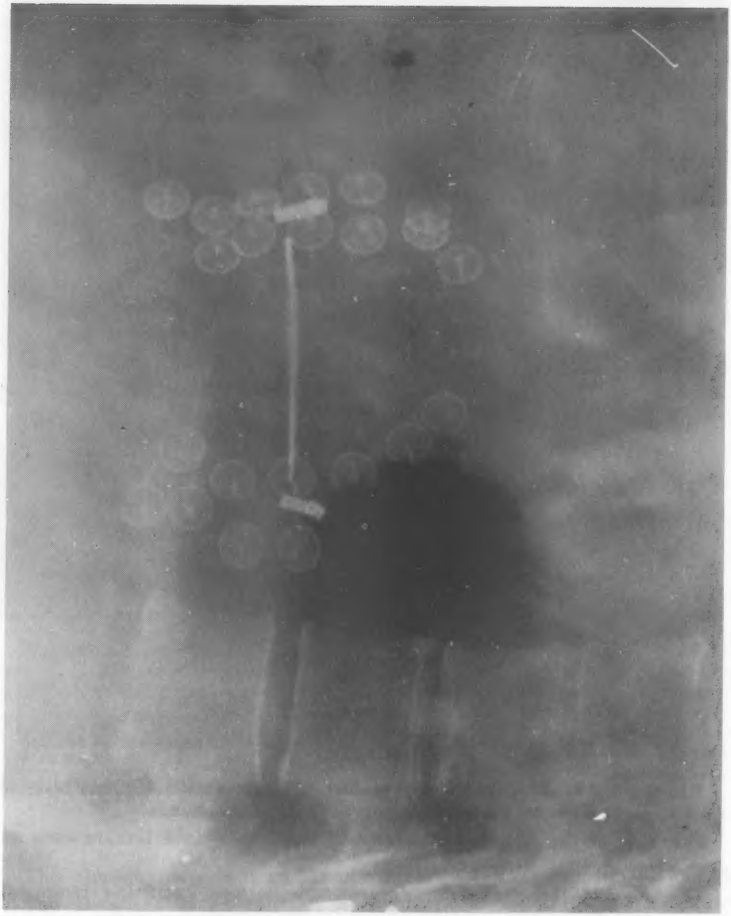

FIGURE 7. Iridium-192 implant of tumor of the chest wall. Four glass dosimeters mounted in tandem in individual gold jackets, located in the mid-plane of a two-plane implant.

Consideration is also being given to *in vivo* survey sets and to the construction of semiconductor mosaics as a replacement for scintillation scanners.

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DIVISION OF ANTHROPOLOGY

KINSHIP ORGANIZATION AMONG THE URBAN BATAK OF SUMATRA*

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This paper outlines the social system of the Toba Batak of the interior mountain villages of North Sumatra, delineates the extent to which the system has changed among the Westernized urban Batak who have migrated to the modern coastal city of Medan, and discusses the implications of the changes that have occurred. Our purpose in describing the organization of village society is to establish a base line for understanding Batak kinship organization in the city. The method of controlled comparison enables us to make fairly firm statements about the consequences of urbanization.

Village Social Organization[1,2]

The Toba Batak are a Malayo-Polynesian-speaking people with a total population of almost one million. Their society is segmented into hundreds of clans and more than one thousand localized exogamous lineages. The localized lineage, the major social group in Batak society, is based upon patrilocal residence and patrilineal descent and inheritance. The male members of each lineage with their spouses live together in their own village, form a ceremonial unit, and exercise corporate control over the terraced rice fields, the most important productive property in the Batak highlands.

Prior to Western contact in the latter part of the Nineteenth Century, each village was relatively autonomous, intervillage warfare was frequent, and no centralized political authority existed. Whenever a lineage became too large to exploit the economic resources available to it, a patrilineal segment would separate from the parent line and would establish itself in a new village. As the population increased, there was further segmentation leading to the formation of numerous hamlets *(huta)*. The village in which we lived (Lintong ni Huta, Tampahan, Balige) contained 219 nuclear family households located in 38 different hamlets, approximately 6 households per hamlet.

To some extent, social bonds are scattered across the lineage landscape. For example, not all of the 219 families in our area were direct descendants of the ancestor who founded the village 8 generations ago. Of those who were not, 22 per cent were descendants of men of related clans *(marga)* who had been invited, in the past, to join the new village

* This paper was presented at a meeting of the Division on November 23, 1959. The data upon which it is based were gathered in Indonesia in 1957-1958. Financial support was obtained from the Ford Foundation, New York, N.Y., the National Science Foundation, Washington, D.C., and Yale University. Helpful suggestions were received from A. R. Diebold, Jr., G. P. Murdock, and D. Schneider.

in order to increase its population, and 12 per cent were the result of unions in which residence after marriage had been matrilocal.

Localized lineages are further linked to one another through genealogical ties, providing a vertical integration of society based upon descent. In native ideology all members of the society are descended from one man, Si Radja Batak, and hence form one super patrilineage. Fission of the agnatic line that began among the sons of the first Batak has continued to the present. As such, genealogical knowledge is of the utmost importance. Individuals of different lineages are always able to determine their proper kinship relationship by tracing their social distance from a common male ancestor. Every man becomes familiar with the genealogy of his own lineage, and some specialists have published genealogical books in the Batak language. One such document[3] of 558 pages consists primarily of the names of the male descendants of one ancestor who lived nineteen generations ago.

It would be erroneous to conceive of Batak society as consisting of a hierarchy of separate descent groups from patrimoieties, groupings of clans, and clans, to maximal and minimal lineages. The localized lineage is a meaningful corporate group, but the higher level patrilineal segments are merely descent categories. The patrimoieties have no apparent function in daily life and, while the members of a clan share a common name and are usually an exogamous unit, they own no property in common, have no political leader, and reside in noncontiguous areas. It is more in accord with the data to conceive of a flexible lineage principle in which one moves up and down the genealogical line creating different groupings of relatives for different social purposes. In life-crises rites such as a funeral, the unilineal structure of the society is symbolized by announcing the names of the male relatives of the deceased on every generation level. As a name is called the descendants of that person who are present form a group, dance together, and receive a particular portion *(djambar)* of the animal slaughtered for the feast.

Because the localized lineages are rigidly exogamous, they are also linked to one another through affinal ties, thus providing a horizontal integration of society. Among the Batak at least three localized lineages are related as an alliance group: one's own lineage, the lineage to which one has given wives *(boru)*, and the lineage from which one has received wives *(hulahula)*. Many more than three lineages are always involved in alliance group relations. From the point of view of any male ego, the men of his wife's lineage are his main *hulahula*, to whom he must show respect, but he also generalizes the relationship to the lineages from which his father, brothers, and other lineage mates have taken wives. It is the same with ego's *boru* lineages whose members have married the sisters and daughters of his lineage. Relationships between the lineages in an alliance group are solidified by matrilateral cross-cousin marriage and the unilateral exchange of goods and services.

Batak alliance groups and the marriage rules upon which they are based would seem to limit societal integration, as a man who marries his mother's brother's daughter takes a wife from the same family as did his father, thereby intensifying an existing relationship.[4] However, the frequency of Batak marriage to a real mother's brother's daughter is only 2.3 per cent. The system would also be restrictive if three or more lineages married in a closed ring, that is, if the men of lineage A of clan I received wives from lineage B of clan II, who received wives from lineage C of clan III who, in turn, received wives from lineage A of clan I, thereby completing the circle. However, according to my census data, the spouses of 140 men within one localized lineage were members of 38 different clans.

The Batak marriage system is quite flexible in practice. Efforts are made to arrange at least one marriage in every generation between members of the lineages in an alliance group who are real or classificatory matrilateral cross-cousins *(pariban)*. If a suitable spouse is not found in the *pariban* class narrowly defined, for example, a female of ego's mother's or father's mother's lineage, then the definition of the class is extended from lineage to clan – a man marries a woman of lineage X rather than lineage Y of clan 1. Even with this extension, in our village the frequency of marriage to a female of ego's mother's clan was only 15 per cent and to ego's father's mother's clan only 3.8 per cent. The Batak do not expect that every man should or will marry his cross-cousin. Every marriage does, however, give rise to a formal relationship between the lineages involved. The relationship may or may not be maintained in future generations by subsequent *pariban* marriage. It will not be maintained if the population of one lineage is severely depleted, if one lineage suffers a series of economic losses, or if the women of the *boru* lineage do not bear many sons.

The net effect is a rather wide range of affinal ties, as the number of relatives one has and the frequency of matrilateral cross-cousin marriage vary in inverse proportion. The Batak seem to have maintained the system of alliance groups, consisting of social, economic, and political relations between lineages, with a comparatively low frequency of marriage to a real or classificatory cross-cousin.[5-7] Although precise data are not available, it is highly probable that the frequency of cross-cousin marriage was higher in the period before Western contact. There is no doubt that even within the last decade village social organization has been subject to severe pressures for change.[8]

Thus in the villages the social system is based upon scattered localized lineages integrated vertically through descent lines and horizontally through affinal ties, providing a kinship grid covering the entire society. However, at the higher levels of segmentation and at the wider extensions of alliance group relations, the bonds are rather weak. The sense of group identity and social solidarity among the village Toba Batak is not highly developed.

Urban Social Organization

The urban Batak located in the city of Medan are exposed to the full impact of Western influence. Medan is a true urban center, the commercial and administrative capital of North Sumatra, with a population of over one third of a million. Within the city there were 4 universities, 9 foreign consulates, 16 movie houses, and more than 50 resident doctors in 1957. The Medan telephone book contained 224 pages. Malay, Javanese, Minangkabau, Atjehnese, and representatives of many other Indonesian peoples in addition to Chinese, Indians, and Europeans live together in the city. The Toba Batak community in Medan was established about 50 years ago, as individuals and families migrated from the highlands to seek employment and a higher standard of living. Let us examine local, descent, and alliance groups among the urban Batak.

The localized lineage no longer exists, as individual families reside in widely scattered households in different parts of the city. One's next-door neighbor might be Chinese, with a Minangkabau family living nearby and another Toba Batak of different clan affiliation across the street. The Chinese, Indians, and Europeans cluster in certain sections of the city, and many Toba Batak reside in one area, Medan Baru, but there is no pattern of residential proximity among lineagemates.

The urban Batak, however, form neighborhood associations based entirely upon residential criteria that crosscut descent categories. All Batak within one defined area – by analogy between 72nd Street and 79th Street and Central Park West and Riverside Drive in New York, N.Y. – belong to a corporate mutual aid society called the *dongan sahuta*, the hamlet group. In most areas members of other Indonesian ethnic groups are excluded. The society has officers, regular dues, and meetings. Members assist one another in times of misfortune and in the performance of life-crises rites. In some sections of Medan the Batak living on one city block form a similar but smaller society, the *dongan sastraat*, the street group. These social groups, *dongan sahuta* and *dongan sastraat*, correspond to village and hamlet in the highlands. However, as each urban family maintains close ties with its original localized lineage in addition to joining the new neighborhood associations, it is actually affiliated with two different sets of local groups, one in the village and one in the city.

The urban Batak also form clan associations, *dongan samarga*. Every Batak in Medan, irrespective of where he may live, has the privilege of joining his clan group if he pays the Rp. 20* initiation fee and the monthly dues of Rp. 2.50. Some associations have established scholarships and a revolving loan fund with low interest rates, almost all have annual gatherings and youth groups, and serve as a mutual assistance and welfare society, but their major manifest function is to aid in the

*In 1958 about 65 cents at the official rate of exchange and about 25 cents at the unofficial rate. The cost of membership varies among different associations.

organization and performance of adat ceremonies in which they occupy a central position. At weddings, funerals, and other rituals the elected head of the association receives on behalf of all members a part of the slaughtered animal *(djambar)*, a symbolic representation of community recognition of the *dongan samarga* as a patrilineal descent group.

As only those living in Medan may join the clan association, its composition is determined by criteria of residence as well as descent and, in this respect, it is similar to the localized lineage. One large association is divided into four sections corresponding to the four geographical areas of Medan. Each section has its own officers, is considered as one segment of the larger group, and recruits its membership from those in the clan who reside in its particular area of the city.

There are important differences between the *dongan samarga* and the localized lineage. The former is like a voluntary association in that no one is obligated to join, although most urban Batak do. In the highlands the clan is a descent category; in the city it becomes a corporate social group. This is the reverse of segmentation and illustrates the flexibility of the Batak lineage system. In some cases two or three related clans that have a common male ancestor will band together to form one association that bears the name of that ancestor. The patrilineal descent groups function on a high level of organization in the city, thus changing the basis of reckoning kinship relationships. When two Batak in different lineages of the same clan meet for the first time in the highlands, one asks, what is your village and who was your grandfather? In Medan one says, I am eighteen generations removed from the founder of our clan, what is your generation level?

A married couple in Medan may join *two* urban clan associations, but in the highlands they reside in only *one* localized lineage. The urban associations include both male and female members of the clan with their spouses; thus a couple in Medan belong to the husband's clan group, which includes his sisters, and to the wife's clan group, which includes her brothers. On the other hand, a localized lineage is composed of a line of men with their wives plus those female members of the lineage with their husbands who reside matrilocally. The vast majority (88 per cent) of the women live in their husband's village, work his rice fields, participate in ceremonies conducted by his lineage, and return to their village of origin only on special occasions. In effect, then, the villagers participate in only one social and ceremonial group, while urbanites participate in at least two. Group affiliation is extended further as most urban families participate in the ceremonies conducted by their hamlet group and localized lineage in the highlands.

Alliance group relations have been maintained in Medan and operate effectively to further social cohesion. Lineages related by affinal ties continue to exchange goods and services. At every life-crisis ceremony performed in the city, representatives must be present from one's own

lineage and the lineages to which one has given and from which one has received wives. A man must show respect and pay homage to the male members of his wife's lineage, irrespective of differences that may exist in wealth or social class position.

A discernible shift is apparent in the attitude of some urbanites toward matrilateral cross-cousin marriage. Many are now opposed to the practice. The reasons given in opposition to *pariban* marriages may be classified in three categories – biological, psychological, and sociological. First, it is felt that the offspring of *pariban* marriage may be mentally defective. Educated Batak are aware of the possibility of deleterious genetic consequences due to inbreeding. Second, unmarried boys claim that they find no challenge in courting their *pariban*, as they have rights to the girl. The peer group teases a boy who contemplates *pariban* marriage, and a few have confided that they regard their *pariban* as if she were a sister. This psychological manifestation of a social change suggests a possible blurring of the distinction between cross and parallel cousins. Third, many Batak argue that it is not socially or economically advantageous for those residing in the city to marry a *pariban*. Today it is considered preferable to take a wife from an unrelated lineage, thereby creating ties with a new group of relatives and establishing contacts that could be helpful in business.

The latter sociological explanation would seem to be crucial in accounting for the resistance to matrilateral cross-cousin marriage. Opposition to cousin marriage is also found in contemporary villages, but it has been greatly intensified by the conditions of urban life. The radical increase in the number of different lineages and clans in Medan has made it more advantageous to form new affinal relationships than to solidify old ones by cousin marriage. As the urban Batak community remains kinship-oriented, the more relatives one has the more secure is one's position. To say the same thing in a different way, marriage bonds are primarily political bonds between lineages. The important factor in the selection of Batak marriage partners, in village and city, has always been the quest for wealth and power. A political alliance created by the exchange of women among a small number of lineages located within a limited area is no longer as meaningful in the highlands as it was a century ago, and is even less meaningful in the city. Members of an urban lineage now attempt to widen the scope of their relationships by establishing affinal ties with as many different kin groups as possible, especially with those that are large and include wealthy merchants and high-ranking government officials.

Conclusion

One consequence of urbanization in other areas of the world has been a diminishing importance of kinship in social life. We have found that social relationships among the Toba Batak community in Medan are

based upon kinship as well as territorial organization, that the unilineal descent group has been retained, and that the village social system has not changed in basic structure when transplanted to the city. These findings do not conform to current views on the evolution of social organization.

Although it would be beyond the scope of this paper to discuss the specific conditions of urbanization in Sumatra that account for our findings,[9] it may be suggested that the Batak social system has been maintained in the city partly because it has been sufficiently flexible to reorganize and recombine pre-existing elements. The structure of the system has not changed radically in Medan in response to the increase in the number of kin groups, the greater population density, and the new conditions of urban life. Rather, the system has adjusted itself to function at a higher level of organization and to provide a wider basis of societal integration. Social ties extend more widely in Medan throughout all segments of the community, binding the individuals so linked into one kinship network. The wider extension of kinship ties has been accomplished at some sacrifice to social solidarity.[10] More people are bound together by less intensive relationships, and they interact less frequently. In-group feeling is stronger among the members of a localized lineage than among those affiliated with an urban clan association; however, the sense of Toba Batak ethnic identity is stronger in the city than in the highlands.

In his review of unilineal descent groups in Africa, Fortes[11] refers to the flexibility of primitive social structures that remain stable in the long run without being rigid. Although unable to define a "flexible" social structure, I see no inherent reason why the emergence of a modern economy requires a Western European family organization nor why lineage and clan cannot serve the same functions as do voluntary associations.[12] Possibly patrilineal systems are better able to survive in some urban environments than matrilineal or even bilateral ones.

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DIVISION OF OCEANOGRAPHY AND METEOROLOGY

A STUDY OF THE EFFECT OF WEATHER ON MORTALITY*

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The meteorologist's interest in the relationships between weather and health dates back to the dawn of meteorology. The physician's interest dates back even farther: at least as far back as Hippocrates and his *Airs, Waters and Places.*[1]

Physicians were early to recognize the need for systematically recorded weather observations in order that the relationship between illness, disease, and weather might be raised above the realm of conjecture. It is not surprising, then, that the first systematic climatic survey of the United States was undertaken on the initiative of the medical profession. This forward-looking step[2] was taken by Joseph Lovell, the Surgeon General of the Army when, in 1818, he ordered the surgeons of the Army to keep detailed day-to-day weather records at all posts. The statistical information collected at these posts was first published and discussed in 1842.[3]

European meteorologists and physicians alike have been prolific in their writings and studies of weather and health and have maintained an active interest in the subject. This is particularly true with reference to studies of meteorological parameters in health resort areas. In the United States, Petersen[4] has presented comprehensive and detailed studies of weather, health, and disease. The meteorologists' initial interests in the subject subsequently subsided to some extent, but they now show definite signs of revitalization. This new impetus apparently stems from three very important factors. First, present-day meteorologists tend more and more to realize that the atmosphere is an environment for living things in addition to being a particularly interesting heat engine; second, there is a growing cognizance and appreciation of the utility and power of the statistical approach; third, the availability of high-speed electronic computers now permits data processing and testing on a scale that heretofore has been prohibitive.

The ultimate approach to studies of the effects of weather on health is the detailed approach: the study and correlation by specific diseases.

*This paper, illustrated with slides, was presented at a meeting of the Division on November 24, 1959. The material in this article is a partial report of graduate work undertaken during an assignment to Rutgers University, New Brunswick, N.J., during the summer of 1959.

The Division of Mycology held a meeting on November 20, 1959 at which Lalla Iverson of the Armed Forces Institute of Pathology, Washington, D.C., presented a paper entitled "Cryptococcosis: Host and Strain Factors." This paper will appear in a later edition of the *Transactions*.

Case histories in sufficient numbers, however, are difficult to obtain. First, the attending physician's permission must be obtained in order to attain access to the medical records; second, a detailed analysis of each case is required in order to decide into which of various populations (in the statistical sense) each should be classed. Even then, numerous borderline cases are encountered.

The approach undertaken here is to use the much grosser concept of death. This approach, through deaths rather than through specific causal factors and "cases," avoids the diagnostic factors that in many instances are far too subjective to lend themselves to rigorous statistical treatment, other than by sheer weight of numbers in a really large sample. By contrast with disease and cause of death, death itself is unique and incontrovertible. The raw data are comparatively easy to obtain, since the offices of vital statistics in all large cities of the United States maintain mortality totals of one sort or another.

If weather has any real effect on mortality, the northeastern area of the United States is a fertile region for such a study. It is in the path of the major storm tracks of the North American continent. As Biel[5] points out, it undergoes a monsoonal change from the domination of cold, dry, polar air in the winter to warm, moist, tropical maritime air in the summer. There are frequent clashes between these two extremes in air masses, and the area is subjected to the sometimes violent storms that accompany such changes.

In June of 1949, the Bureau of Records and Statistics of the city of New York, N.Y., added to its previous procedure a detailed tabulation of mortality by actual date of occurrence (previous records, as is generally the case, were tabulated on the basis of date of report) and for each of seven age groups. Beginning in June 1952, the tabulations were categorized further into white and nonwhite groups. It is therefore now possible, for the first time, to make a detailed study of the correlation of meteorological parameters in a really active weather regime with detailed mortality statistics.

These mortality data were first analyzed to determine whether there were any marked singularities or anomalies that should either be corrected or studied further. TABLE 1, dealing with average weekday mortality, shows a maximum on Monday for most age groups and a minimum on Sunday. The Monday maxima of 227.4 for total mortality and 77.6 for the 45 to 64 year age group are both statistically significant at the 3 per cent level (in other words, there is only a 3 per cent possibility that this difference could have occurred purely by chance). Since no corresponding 7-day periodicity has been determined from the weather records, it is concluded that the explanation is inherent in the weekly habits of the population rather than being due to any direct causal weather realtionship; the cause is apparently sociological rather than meteorological.

TABLE 1

AVERAGE WEEKDAY MORTALITY

Age Group	*Sun.*	*Mon.*	*Tues.*	*Wed.*	*Thurs.*	*Fri.*	*Sat.*
1	10.8	11.0	11.1	10.9	11.5	10.9	11.2
1 to 4	1.6	1.8	1.8	1.6	1.7	1.8	1.8
5 to 14	1.3	1.4	1.4	1.4	1.5	1.4	1.4
15 to 24	2.3	2.3	2.1	2.3	2.3	2.3	2.5
25 to 44	16.2	16.7	16.5	16.2	16.4	16.4	16.8
45 to 64	72.9	77.6	73.8	74.0	73.8	73.7	74.1
≥65	114.1	116.6	115.1	114.5	114.7	114.7	113.3
Total	219.1	227.4	221.8	220.9	221.8	221.1	221.0

Neither the Sunday minima nor the other Monday maxima are significant at the 5 per cent level generally taken as the test for statistical significance. For the purpose of this study, therefore, no weekday correction factors were used, even for the two instances with 3 per cent significance.

The period of highest over-all mortality in the 9½ years of record was that for the Asian influenza pandemic that lasted from October 1957 through March 1958. Comparison of total mortality with temperature and precipitation in the city of New York during these and antecedent months showed no significant correlation. Similar comparisons of mortality with local weather for other influenza months, determined on the basis of records and publications available in the New York Bureau of Records and Statistics, gave similar results.

With reference to the Asian influenza pandemic of 1957-1958, the "Introduction" to the *Bulletin of the World Health Organization*[6] states: "The pandemic began in China, probably at the beginning of the spring of 1957... It was carefully followed as it spread across the world, and in some countries its arrival was even predicted and awaited."

Small wonder, then, that local weather in the metropolitan area of New York was no precursor. For a study of the effects of weather on influenza, case histories and the segregation of deaths by causes are required. The segregation of deaths by causes as listed on death certificates is generally not encouraged because of the diagnostic factors involved. As indicated earlier, case histories are not readily obtained. It was therefore decided that epidemics of influenza and other respiratory diseases would be considered outside the population of this particular study. Except for the data given in TABLES 1 and 4, data for "influenza periods" have been omitted accordingly.

The meteorological data used in this study are those from observations taken at the United States Weather Bureau's observatory in Central Park in New York, N.Y. Of the five sources of observational data available

TABLE 2

REGRESSION ANALYSES TESTED ON 1959 DATA

		25 to 44 year old			45 to 64 year old		
Month	*Temp.*	*Obs.*	*Calc.*	*Error*	*Obs.*	*Calc.*	*Error*
January	31.1	430	511	+3.1	2347	2457	+1.2
June	71.2	435	493	+1.2	2067	2163	+0.7
July	76.3	435	484	+0.8	2024	2083	+0.7
August	77.5	480	482	+0.03	2106	2059	−0.7

		65 and over			Total mortality		
Month	*Temp.*	*Obs.*	*Calc.*	*Error*	*Obs.*	*Calc.*	*Error*
January	31.1	4449	4016	−2.6	7846	7488	−5.3
June	71.2	3716	3327	−1.2	6787	6484	−1.7
July	76.3	3484	3149	−0.9	6448	6190	−0.9
August	77.5	3889	3007	−3.6	7009	6090	−5.2

Key: Obs. = Actual mortality; Calc. = Mortality calculated from regression equation;

$$\text{Error} = \frac{\text{observed} - \text{calculated}}{\text{standard error}}$$

in the area, these are considered more nearly representative of the average environmental conditions of the population as a whole.

TABLE 3

CORRELATION COEFFICIENTS

Monthly Mortality on Monthly Average Temperature

		Age Group							
		25 to 44		*45 to 64*		*≥ 65*		*Total*	
Month	*d.f.*	*r*	*%*	*r*	*%*	*r*	*%*	*r*	*%*
Jan.	8	0.859	1	0.509	–	-0.933	0.1	-0.958	0.1
May	6	-0.152	–	-0.278	–	-0.234	–	–	–
June	9	0.196	–	0.583	6	0.257	–	0.670	5
July	9	0.317	–	0.581	6	0.384	–	0.673	5
Aug.	9	0.206	–	0.306	–	0.144	–	0.541	–
Dec.	8	0.342	–	0.232	–	-0.568	7	-0.740	2

Key: d.f. = degrees of freedom; r = correlation coefficient; % = level of significance ("–" = ≤ 10%).

TABLE 4

PRONOUNCED FRONTS AND TOTAL MORTALITY

		Average mortality		Number of months		
Month	*N*	*W*	*W/O*	*W**	*W/O**	*None*
Jan.	52	244.3	243.8	7	1	1
Feb.	39	247.4	237.4	7	1	1
Mar.	48	245.0	237.0	5	2	2
Apr.	52	230.2	227.1	6	3	0
May	52	222.7	214.2	5	4	0
June	49	229.3	215.5	8	1	0
July	41	198.4	201.9	5	3	2
Aug.	36	194.5	189.6	7	2	1
Sept.	57	203.9	203.5	5	5	0
Oct.	49	211.4	218.6	1	8	1
Nov.	55	229.6	224.3	8	2	0
Dec.	57	237.9	236.7	6	4	0
Total	587			70	36	8

N = Number of days with passage of one or more pronounced fronts.
W = Average total mortality on days with pronounced fronts.
W/O = Average total mortality on days without pronounced fronts.
W* = Number of months with maximum mortality average on days with fronts.
W/O* = Number of months with maximum mortality average on days without fronts.

Gordon and Erhardt[7] have referred to "the general inverse relationship of temperature and death" and have shown this with 1957 data for the city of New York. The same general relationship has been shown in curves and data for particular diseases both here and abroad by other writers.[8,9] FIGURE 1 shows annual temperature and mortality curves for New York, N.Y., Cincinnati, Ohio, and Los Angeles, Calif. The temperature curves are based on 1921 to 1950 monthly normal temperatures; the mortality curves are based on daily average mortality for each month, computed from 1949 to 1958 monthly totals of mortality reported for all age groups. The same general relationship is apparent for all three cities.

The solid-line curves of FIGURE 2 give a breakdown by age groups. The general seasonal trend of high wintertime and low summertime mortality is apparent in the infant group, disappears entirely for about 24 years, then manifests itself again, reaching its maximum in the 65-year-and-over age group. A comparison with birth rate statistics for New York for this same period shows no significant seasonal variation that would permit one to consider this seasonal trend in infant mortality a result of a seasonal fluctuation in the infant population. This apparent response to seasonal climatic change among infants could well be an

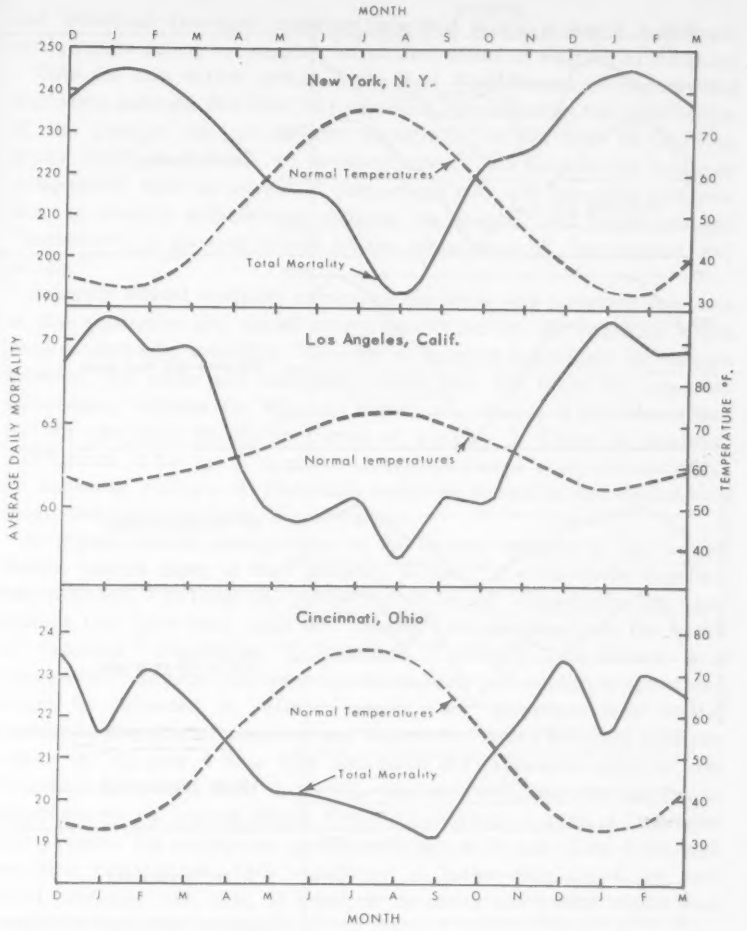

FIGURE 1. Annual mortality curves for New York, N.Y., Los Angeles, Calif., and Cincinnati, Ohio.

indirect effect due to colds and other infectious ailments passed along to the infant by its parents and others. This would account for the disappearance of the seasonal trend after infancy.

The dotted curve in the top panel of FIGURE 2 was prepared from data given by Huntington[10] for the years 1900 to 1911. This shows a double seasonal maximum, the second appearing in July. This summertime maximum is evidence of the *Pseudosaisonkrankheiten* (pseudoseasonal illnesses) mentioned by De Rudder.[11] It was due to a maximum of stomach

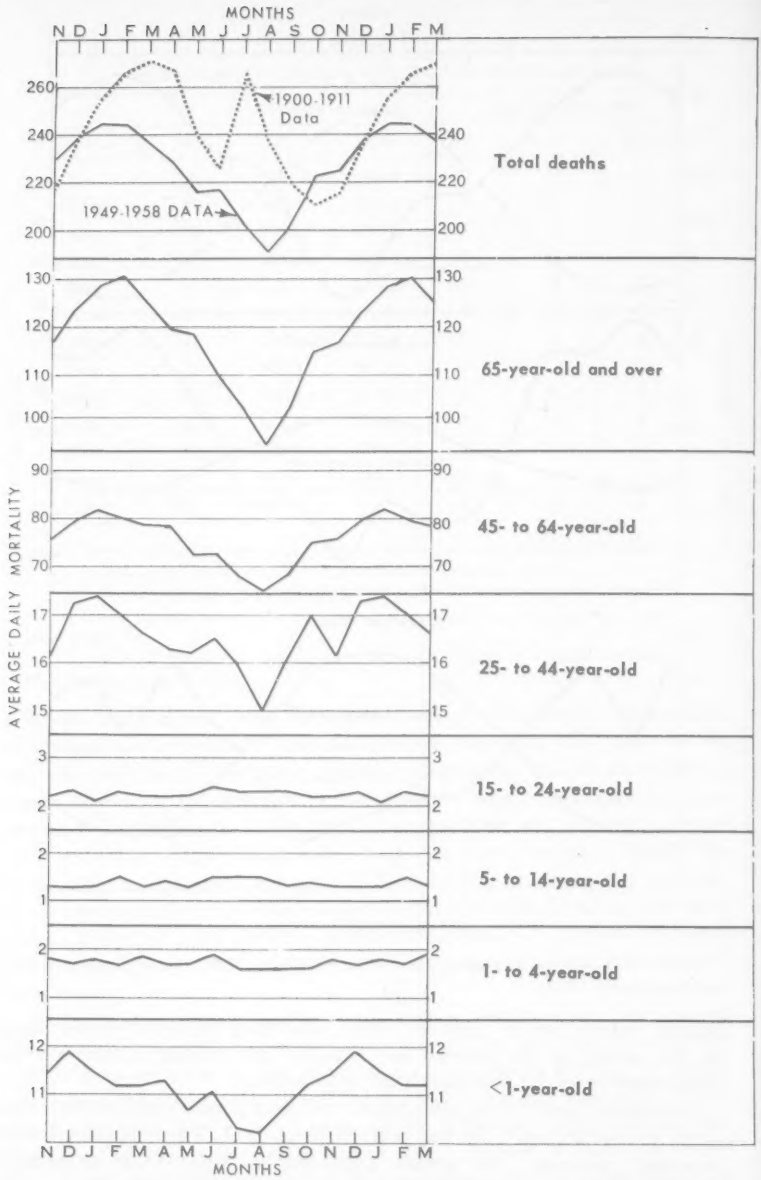

FIGURE 2. Annual mortality curves for all age groups. Solid lines are 1949-1958 data. Dotted line is 1900-1911 data.

and intestinal disorders resulting from food spoilage due to inadequate refrigeration during hot weather: an indirect effect of weather on mortality.

Data for this earlier period in the New York Bureau of Records and Statistics indicate that this July maximum was primarily the contribution of the younger age groups and, especially, of the one- to four-year group, which now shows no seasonal trend. This summertime maximum disappeared with the advent of pasteurized milk and adequate refrigeration as electric refrigerators replaced the familiar and inadequate old "iceboxes": a glowing tribute to the advantages of "our modern way of life."

Separate annual mortality curves for the white and nonwhite segments of the population are shown in FIGURE 3 for the 6½ years for which such records were available. There are no apparent significant differences between the white and nonwhite curves, nor are there any apparent differences between the 6½ year curves of FIGURE 3 for whites and the 9½ year total population curves of FIGURE 2. There is, however, a difference in the age of maximum mortality between white and nonwhite, as shown in FIGURE 3. The white maximum occurs in the 65-and-over group, the nonwhite in the 45 to 64 group.

A popular belief among many of the earlier settlers in the United States, passed along to their progeny, is that "a warm winter begets a full cemetery." Is there any basis for this belief? Considering the many studies that have been made on "comfort" temperatures and the works of Petersen,[4] Huntington,[10] Schickele,[12] Mills,[13] and others, it is reasonable to suspect that extremes in seasonal and monthly temperatures would be reflected in perhaps proportionate departures from normal mortality. The scatter diagrams and regression curves for total mortality shown in FIGURE 4 bear this out. Each dot represents data for one noninfluenza month: data that were used in developing the regression equations for the curves shown. For the two winter months of December and January, the correlation coefficients are -0.74 and -0.96. Such high negative correlations, both significant at better than the 2 per cent level, indicate that now, at least, it is really the colder winter that results in the fuller cemetery.

Except for August, all of the correlation coefficients indicated in FIGURE 4 are significant at the 5 per cent level or better. For the spring and fall months the correlations are insignificant and meaningless. This is to be expected as the correlation swings twice through zero each year in its annual course from significant negative wintertime values to significant positive summertime values.

Data thus far available for 1959 provide the first opportunity to check, with independent data, the regression equations for the curves in FIGURE 4. The *X*s in the diagram are the 1959 data. TABLE 2 gives the actual temperatures and mortality for January, June, July, and August of 1959 for those age groups that had shown seasonal trends. Calculated

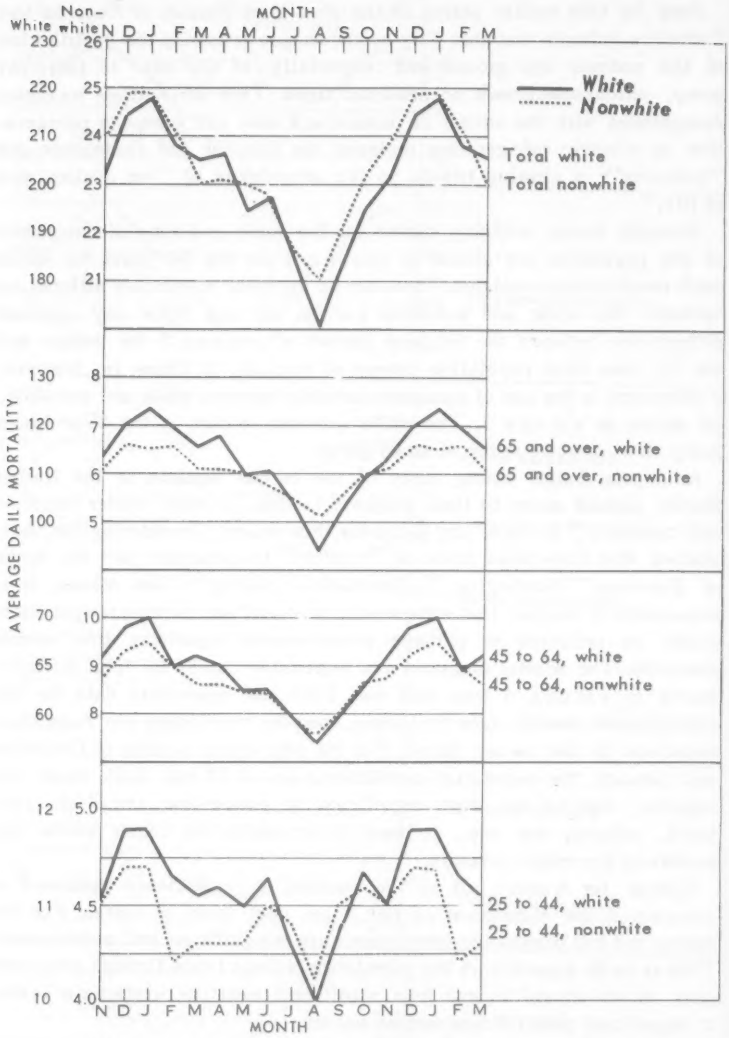

FIGURE 3. Annual mortality curves for white and nonwhite, excluding influenza months.

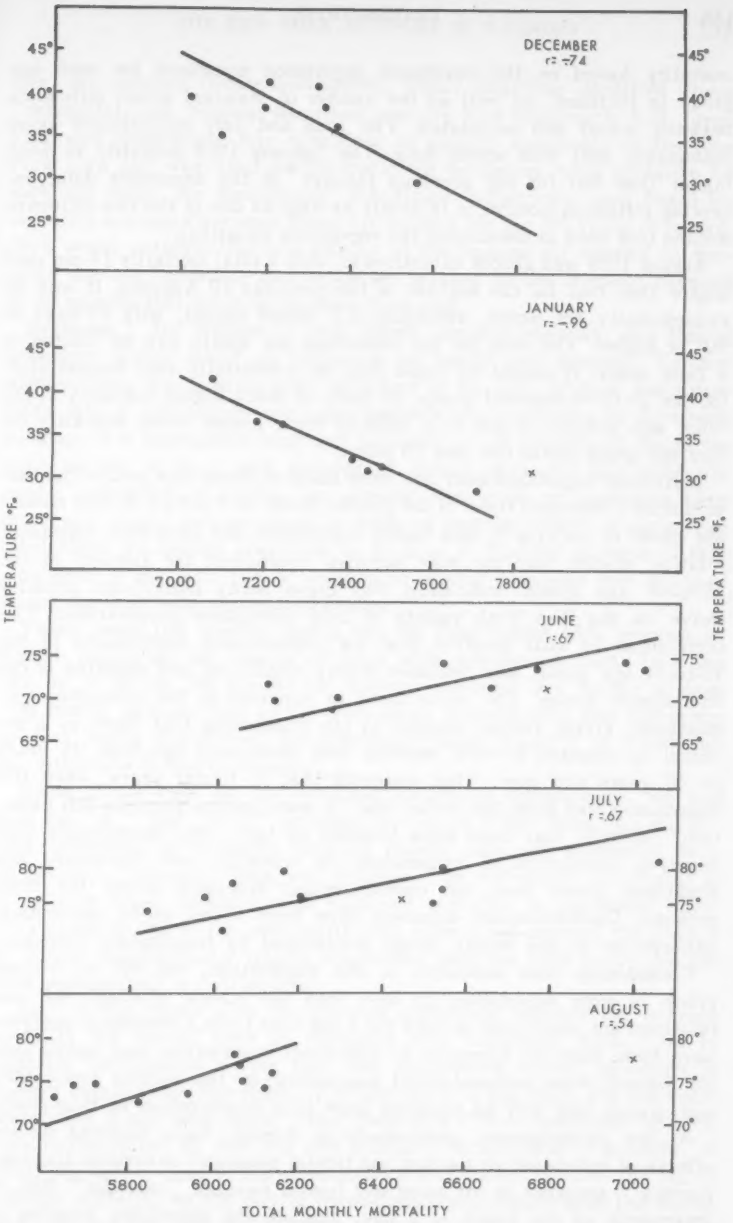

FIGURE 4. Regression of total monthly mortality on monthly average temperature.

mortality based on the developed regression equations for each age group is included, as well as the number of standard errors difference between actual and calculated. The June and July calculations agree reasonably well with actual data. The January 1959 mortality is much higher than that for any previous January* in the dependent data, excepting influenza months; it is nearly as high as one of the two influenza months (not used in developing the regression equation).

August 1959 was almost catastrophic, with a total mortality 14 per cent higher than that for the highest of the previous 10 Augusts. It was an exceptionally hot month, averaging 3.2° above normal, with 13 days of 90° or higher. The data for the individual hot spells will be treated in a later study. It should be noted that the essentially zero August error for the 25-to-44-year-old group, in spite of much higher mortality in the other age groups, is due to a definite trend toward lower mortality for this age group during the past 10 years.

Individual regression analyses were made on those age groups that had exhibited a seasonal trend in the graphs shown in FIGURE 2. The results are given in TABLE 3. One highly significant and somewhat surprising relation stands out: the high *positive* coefficient for January in the 25-to-44 age group, indicating that these hardy individuals actually thrive on the New York variety of cold wintertime temperatures. The correlation is still positive (but not statistically significant) in the 45-to-64 age group, then becomes highly significant and negative in the 65-and-over group. The same trend is apparent in the December correlations, giving further support to the conclusion that there is a reversal in reaction to cold weather with increasing age from 25 years to 65 years and over. This suggests that in former years, when life expectancy was less, the belief that "a warm winter begets a full cemetery" actually may have been founded on fact. The increasingly high negative correlation of temperature on mortality with increasing age doubtless stems from the cardiovascular diseases among the older persons. Cardiovascular diseases have been shown to be particularly susceptible to the bodily strain occasioned by temperature extremes.

Considering heat tolerance in the summertime, the 45- to 64-year group is more susceptible to heat than the others, although the correlations are significant at only the 6 per cent level. Correlation analyses have been made for tolerance to individual summertime heat spells (and for several other meteorological parameters) by the various susceptible age groups and will be reported later in a continuation of this study.

A few investigators, particularly in Europe, have reported on the effects of individual air masses and frontal passages on various diseases. Havens[14] prepared an air mass and frontal passage "calendar" that, in increments of one fourth of a day, indicate the day-to-day duration of

*It has been pointed out since that practically all of 1959 thus far has been a period of unusually high mortality in the city of New York.

four air masses (Polar Continental, Polar Maritime, Modified Polar Continental, and Tropical Maritime) over the New Brunswick, N.H., area. With rare exceptions, this tabulation is equally applicable to the New York area and was used as the basis for tabulating total mortality for each air mass. No statistically significant differences were found for mortality averages between these air masses for the 9½ years of record. This would indicate that any effects of the various air masses on mortality are not immediate and hence are masked by the frequent changes in air masses that occur in the New York area.

Petersen,[4] De Rudder,[11] and others suggest reasons why frontal passages should exert an influence on health and disease. To test whether this was significantly reflected in the city of New York mortality rate, a comparison was made between total mortality on days with passage of a pronounced front and mortality on all other days. Since the total number of days with pronounced frontal passages was generally small in individual months, no attempt was made to segregate them into cold, warm, and occluded in order to test the effect of each. The tabulations by months are given in TABLE 4. For the purpose of this tabulation a day with two or more weak frontal passages was included among the days with pronounced fronts.

There were 106 months during the 9½ year period with pronounced frontal passages, the number of days with pronounced fronts totalling 587. In 70 of the 106 months the average mortality was higher on days with frontal passage than on all other days. Statistically, this difference is very highly significant, the likelihood being nearly 1 in 1,000 that the difference is due to chance.

Using 5 per cent as the level for statistical significance, we find from the "W*" and "W/O*" columns in TABLE 4 that there is a significant difference in mortality between frontal days and nonfrontal days in January, February, June, and October. The difference in October is in reverse order, however, with maximum mortality on nonfrontal days. This could be due to the possibility that, after a hot summer, the change to cooler weather under frontal conditions (and the cold fronts are the more pronounced) is exhilarating.

June is the month with the greatest difference between the average mortality for frontal days as compared with nonfrontal days. This is partially attributable to the fact that pronounced frontal passages in summer herald either the approach or the end of a hot spell. For hot-spell mortalities it has been found[15] that there is a direct correlation between high temperatures on one day and mortality on the next. Therefore, in about one half or more of the pronounced frontal cases in June, the higher average mortality on the day of frontal passage reflects also the higher mortality from the heat wave of the previous day. Since by far the highest heat wave mortality values during these ten summers occurred in June, the effect is more pronounced for June than for the other summer months.

The conclusions reached thus far in this study for New York indicate:

(1) The seasonal trend in mortality has undergone a change since the beginning of the century; this change was due to elimination of a previous indirect effect of climate on mortality.

(2) The seasonal variation in mortality disappears entirely in the groups between 1 and 24 years of age.

(3) No significant difference is apparent between the seasonal mortality trends of white and nonwhite categories.

(4) Mortality is higher during cold winters and hot summers and lower during warm winters and cool summers.

(5) Whereas mortality among the older age groups increases during cold winters, those hardy individuals between 25 and 44 actually appear to thrive under the influence of colder wintertime temperatures.

(6) There is no noticeable difference in total mortality in one air mass as compared with another air mass.

(7) Mortality is significantly higher on days with pronounced frontal passage as compared with other days.

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CONFERENCES HELD

SECTION OF BIOLOGICAL AND MEDICAL SCIENCES

MODERN CONCEPTS OF THYROID PHYSIOLOGY

Thursday, November 5
Friday, November 6 and
Saturday, November 7, 1959

Conference Chairman: Rulon W. Rawson
Sloan-Kettering Institute for Cancer Research, New York, N. Y.

THURSDAY, NOVEMBER 5, 1959

INTRATHYROIDAL BIOCHEMICAL REACTION

9:00 A.M. –
Greetings from the Academy – Robert L. Kroc, Chairman, Section of Biological and Medical Sciences, The New York Academy of Sciences, New York, N. Y.; Warner-Lambert Research Institute, Morris Plains, N. J.

"Iodide Metabolism in Lower Vertebrates" – Jacques Leloup and Maurice Fontaine, Laboratoire de Physiologie, Muséum d'Histoire Naturelle, Paris, France.

"Concentration and Organic Binding of Radioiodine by the Thyroid Gland" – Seymour H. Wollman, National Institutes of Health, Public Health Service, Bethesda, Md.

"Some Factors that Affect Thyroid Hormone Biosynthesis" – Rosalind Pitt-Rivers, National Institute for Medical Research, London, England.

2:00 P.M. –
"Iodoproteins in the Thyroid" – Jack Robbins, Joseph E. Rall, and Harold Edelhoch, National Institutes of Health, Public Health Service, Bethesda, Md.

"The Thyroid Gland as Source and Target in Autosensitization" – Ernest Witebsky, University of Buffalo School of Medicine, Buffalo, N.Y.

"Deiodination of the Iodinated Amino Acids" – John B. Stanbury, Massachusetts General Hospital, Boston, Mass.

FRIDAY, NOVEMBER 6, 1959

TRANSPORT AND METABOLISM OF THYROID HORMONES

9:00 A.M. –

"The Thyroxine-Binding Proteins of Plasma and Their Interaction with Thyroid Hormones" – Sidney H. Ingbar, Thorndike Memorial Laboratory, Boston City Hospital, Boston, Mass.

"The Peripheral Metabolism of Thyroid Hormones" – Raymond Michel and Jean Roche, Collège de France, Paris, France.

"Biochemical Applications of a Newly Discovered Property of Thyroxine" – J. R. Tata, National Institute for Medical Research, London, England.

CELLULAR MECHANISMS IN ACTIONS OF THYROID HORMONES

2:00 P.M. –

"Thyroxine and the Swelling and Contraction of Mitochondria" – Albert L. Lehninger, Johns Hopkins School of Medicine, Baltimore, Md.

"The Influence of Thyroxine and Related Compounds on Oxidation Rate and Efficiency of Phosphorylation in Liver Mitochondria and Submitochondrial Particles" – J. Ramsey Bronk, Columbia University, New York, N.Y.

"Enzyme Responses to Thyroid Hormones" – Henry Lardy, Institute for Enzyme Research, University of Wisconsin, Madison, Wis.

SATURDAY, NOVEMBER 7, 1959

METABOLIC EFFECTS OF THYROID HORMONES AND THEIR ANALOGUES

9:00 A.M. –

"Comparative Effects of Thyroxine Analogues in Experimental Animals" – William L. Money, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"Thyroxine Antagonism by Partially Iodinated Thyronines and Related Analogues" – S. B. Barker and C. S. Pittman, University of Alabama Medical Center, Birmingham, Ala.

"Metabolic Effects of Thyroxine Analogues in Hyman Myxedema" – Richard S. Benua, Robert D. Leeper, Soichi Kumaoka, and Rulon W. Rawson, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"The Metabolic Effects of Thyroid Analogues in Psychiatric Patients" – Frederick F. Flach, Peter E. Stokes, Edward Liang, Rulon W. Rawson, and Oskar Diethelm, Payne Whitney Clinic and Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"The Effect of Thyroid Hormones on Adrenal Steroid Metabolism" – Gordon M. Tomkins, National Institutes of Health, Public Health Service, Bethesda, Md.

"The Effects of Thyroid Hormones on the Metabolism of Steroids" – Thomas F. Gallagher, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

CHEMISTRY AND PHYSIOLOGY OF THE THYROID-STIMULATING HORMONE

2:00 P.M. –

"The Purification and Chemistry of the Thyroid-Stimulating Hormone" – John G. Pierce, Mary E. Carsten, and Leslie K. Wynston, University of California Medical Center, Los Angeles, Calif.

"Chemical Derivatives of the Thyroid-Stimulating Hormone" – Martin Sonenberg and William L. Money, Sloan-Kettering Institute for Cancer Research, New York, N.Y.

"The Release of TSH from the Pituitary" – Samuel Rose, National Institutes of Health, Public Health Service, Bethesda, Md.

"The Participation of the Nervous System in the Control of Thyroid Function" – Monte A. Greer, Takashi Yamada, and Shiro Iino, University of Oregon Medical School, Portland, Ore.

Robert L. Kroc
Chairman

Charles Noback
Vice-Chairman

CARE AND DISEASES OF THE RESEARCH MONKEY

Thursday, November 19 and
Friday, November 20, 1959

Conference Chairman: Robert M. Sauer
School of Veterinary Medicine, University of Pennsylvania,

Thursday, November 19, 1959

Session Chairman: Donald W. Baker
New York State Veterinary College, Cornell University,
Ithaca, N. Y.

9:00 A. M. –
Greetings from the Academy – Robert L. Kroc, Chairman, Section of Biological and Medical Sciences, The New York Academy of Sciences, New York, N. Y.; Warner-Lambert Research Institute, Morris Plains, N. J.

"The Monkey: A Comparison of the Natural Environment with Observations in Captivity" – Frederick A. Ulmer, Jr., Philadelphia Zoological Garden, Philadelphia, Pa.

"Climatic Conditions in the Natural Environment of Monkeys" – Francis K. Davis, Jr., Drexel Institute of Technology, Philadelphia, Pa.

"Problems Associated with the Transportation of Monkeys" – Donald G. DeValois, Eli Lilly and Company, Indianapolis, Ind.

"Colony Husbandry of Research Monkeys" – D. B. Gisler, R. E. Benson, and R. J. Young, The Radiobiology Laboratory of the University of Texas and the United States Air Force, Austin, Tex.

"Care of the Large Monkey Colony" – Paul E. Ayres, Parke Davis & Co., Detroit, Mich.

"Mass Treatment of Monkeys with Antibacterial Substances" – Harry C. Fegley, Merck Sharp & Dohme, Di vision of Merck & Co., Inc., West Point, Pa., and Robert M. Sauer, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

Session Chairman: John T. McGrath
School of Veterinary Medicine, University of Pennsylvania,
Philadelphia, Pa.

2:00 P. M. –
"Studies of the Effects of Brain Lesions on Social Behavior in *Macaca mulatta:* Methodological and Theoretical Considerations" – Allan F.

Mirsky, National Institute of Mental Health, Public Health Service, Bethesda, Md.

"Aging of Monkeys by Dental Formula" – Veikko O. Hurme, Plymouth, N. H.

"Correlation of Skeletal Growth, Epiphyseal Ossification, and Body Weight with Age of Monkeys" – D. B. Gisler, The Radiobiology Laboratory of the University of Texas and the United States Air Force, Austin, Tex., and S. G. Wilson, Jr., Medical School, University of Arkansas, Little Rock, Ark.

"Hematology of the Normal Monkey" – George M. Krise, Radiation Biology Laboratory, Texas Engineering Experiment Station, College Station, Tex.

"Normal Electrocardiogram of the Rhesus Monkey" – Gabriel Atta and Peter W. Vanace, South Jersey Medical Research Foundation, Camden, N.J.

"Spontaneous and Induced Neoplasms of the Monkey" – Sidney P. Kent, The University of Alabama Medical Center, Birmingham, Ala.

Friday, November 20, 1959

Session Chairman: Lewis L. Coriell
South Jersey Medical Research Foundation,
Camden, N. J.

9:00 A. M. –
"Parasitism in Monkeys" – George L. Graham, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

"Influence of Crowding on Monkey Health" – Alan A. Creamer and Ramsey S. Buchanan, Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa.

"The Roles of Infectious and Noninfectious Diseases in Monkey Health" – Robert M. Sauer, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa., and Harry C. Fegley, Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa.

"Oral Diseases of Primates" – D. Walter Cohen, School of Dentistry, University of Pennsylvania, Philadelphia, Pa., and Henry M. Goldman, School of Medicine, Boston University, Boston, Mass.

"Diseases of the Nervous System of Monkeys" – J. R. M. Innes, Brookhaven National Laboratories, Upton, N. Y.

"Experimental Streptococcal Infection in the Rhesus Monkey: Attempts at the Production of Rheumatic Fever" – Peter W. Vanace, South Jersey Medical Research Foundation, Camden, N. J.

Session Chairman: O. S. Nordland
Defence Research, Kingston Laboratory,
Kingston, Ont., Canada

2:00 P. M. –

"Normal Flora and Pathogenic Enteric Bacteria of Monkeys" – Nathan J. Schneider, Florida State Board of Health, Jacksonville, Fla.

"Enteric Viruses of Monkeys" – F. S. Cheever, R. L. Heberling, School of Medicine, University of Pittsburgh, Pittsburgh, Pa.

"A Pox Disease of Monkeys" – J. E. Prier, Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa., and Robert M. Sauer, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

"B-Virus Infection in Monkeys" – Stanley A. Keeble, Glaxo Laboratories, Sefton Park, Stoke Poges, Bucks, England.

"B-Virus Infection in Man" – W. L. Davidson, Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa., and Klaus Hummeler, Childrens Hospital, Philadelphia, Pa.

"Experimental Immunization Against B-Virus" – R. E. MacLeod, Connaught Medical Research Laboratories, University of Toronto, Toronto, Ont., Canada.

Summary – Leonard J. Goss, Cleveland Zoological Society, Cleveland, Ohio.

Robert L. Kroc
Chairman

Charles Noback
Vice-Chairman

DIVISION OF INSTRUMENTATION

AUTOMATIC CHEMICAL ANALYSIS

Thursday, November 12
Friday, November 13 and
Saturday, November 14, 1959

Conference Chairman: Ralph H. Muller
Analytical Chemistry, Santa Fe, N. Mex.

THURSDAY, NOVEMBER 12, 1959

Session Chairman: Leonard T. Skeggs
Western Reserve University, Cleveland, Ohio

9:00 A.M. –
Greetings from the Academy - Duncan A. Holaday, Chairman, Division of Instrumentation, The New York Academy of Sciences, New York, N. Y.; Department of Anesthesiology, The University of Chicago Clinics, Chicago, Ill.

Introduction - Ralph H. Muller, *Analytical Chemistry,* Santa Fe, N. Mex.

"Automation of Enzyme Determinations" -

(1) M. K. Schwartz, Memorial Center for Cancer and Allied Diseases, New York, N. Y.
(2) Gerald Kessler, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.
(3) O. Bodansky, Memorial Center for Cancer and Allied Diseases, New York, N.Y.

"Cholinesterase Activity Determination in an Automated Analysis System" - George D. Winter, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Continuous Automatic Integrated Flame Photometry" - Part I, Instrumentation: Milton Pelavin and J. Isreeli, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.; Part II, Analytical Data: Gerald Kessler, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Determination of Carbon Dioxide in Blood Serum" - Leonard T. Skeggs, Western Reserve University, Cleveland, Ohio.

"Continuous Automatic Determination of Cholesterol" - Gerald Kessler, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

Session Chairman: Leonard T. Skeggs,
Western Reserve University, Cleveland, Ohio.

2:00 P.M. –

"A Random Selection System for Automatic Dynamic Biochemical Analysis by a Time-Sharing Method" - Raymond Jonnard, Prudential Insurance Company of America, Newark, N. J.

"Automatic and Continuous *in Vivo* Organ Perusal Experimentation" -

(1) Andres Ferrari, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.
(2) Gerald Kessler, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.
(3) Frank Russo-Alesi, Squibb Institute for Medical Research, New Brunswick, N. J.
(4) John Poutsiaka *et al.*, Squibb Institute for Medical Research, New Brunswick, N. J.

"The Use of the Autoanalyzer in Studies of the Physiological Chemistry of the Parotid Gland" - Ira L. Shannon, School of Aviation Medicine, USAF Air University, Randolph Air Force Base, Tex.

"Automatic Analysis of Amino Acids" - Kenneth R. Woods and Ralph L. Engles, Jr., Cornell University Medical College, New York, N. Y.

"The Solution of Problems Involving Special Handling of Samples and Reagents in an Automatic Analysis System" - Irvin E. Taylor and Max M. Marsh, Eli Lilly and Co., Indianapolis, Ind.

FRIDAY, NOVEMBER 13, 1959

Session Chairman: Jacques Kelly
Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

9:00 A.M. –

"Automation of the Microbiological Assay of Antibiotics with an Autoanalyzer Instrumental System" -

(1) Joseph Pagano, Squibb Institute for Medical Research, New Brunswick, N. J.
(2) John Gerke, Squibb Institute for Medical Research, New Brunswick, N. J.
(3) Andres Ferrari, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Nitrogen Determination by a Continuous Digestion and Analysis System" - Andres Ferrari and Bernard Searle, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"A Method for the Determination of Ammonia on the Autoanalyzer" E. E. Logsdon, Eli Lilly and Co., Indianapolis, Ind.

"Continuous, Automatic Chemical Analysis of Nitrate in the Presence of Ammonia and Urea" - Eugene Catanzaro, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Industrial Applications of the Titrilop" - Arthur E. Dean, Consolidated Electrodynamics Corp., Pasadena, Calif.

Session Chairman: Jacques Kelly
Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

2:00 P.M. –

"Continuous Sample and Blank Analysis by Means of Differential Colorimetry" - Frank Russo-Alesi, Constance Sherman, and Jacques Kelly, Squibb Institute for Medical Research, New Brunswick, N. J.

"Partial or Complete Automation as Alternatives Available to the Analyst"-D.R.Chapman, Baird and Tatlock (London) Ltd., London, England.

"Recent Developments in Automatic Colorimetric Chemical Analysis Instruments" -
(1) Robert T. Sheen, Milton Roy Company, Philadelphia, Pa.
(2) Earl J. Serfass, Lehigh University, Bethlehem, Pa.

"An Automated Method for Determination of Terramycin" - T. C. Grenfell, D. J. McLaughlin, W. H. McMullen, and Jacques Kelly, Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

"Continuous and Automatic Analysis of Process Streams by Gas Chromatography" - H. J. Maier and H. N. Claudy, Perkin-Elmer Corp., Norwalk, Conn.

SATURDAY, NOVEMBER 14, 1959

Session Chairman: Dwight Gillette
Refined Syrups and Sugars Inc., Yonkers, N. Y.

9:00 A.M. –

"Automated Enzymatic Assay of Organic Phosphate Pesticide Residues" George D. Winter, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Use of the Technicon Autoanalyzer for Determination of Protein, Phosphate, and Reducing Sugars in Yeast Molasses and Grain Work" - George Reinhart and William Hardwick, Central Research Department, Anheuser-Busch Inc., St. Louis, Mo.

"Automatic Enzymatic Analysis for L-Lysine Monohydro-Chloride via Decarboxylation" -

(1) George Schaiberger, E. Merck and Co., Elkton, Va.
(2) Andres Ferrari, Research Laboratories, Technicon Instruments Corp., Chauncey, N. Y.

"Automation of Sugar Analysis in Barometric Condensers and Boiler Waters" - Dwight Gillette and Elliot Baum, Refined Syrups and Sugars, Inc., Yonkers, N. Y.

"Methods Development for Phosphate Analysis with the Autoanalyzer" - Daniel P. Lundgren, Research and Development Division, Lever Brothers Co., Edgewater, N. J.

Session Chairman: T. Finnegan
Niagara Mohawk Power Corp., Buffalo, N. Y.

2:00 P.M. –

"Automatic Analytical Instrumentation in the Modern Power Plant" W. A. Crandell, Consolidated Edison Company of New York, New York, N. Y.

"Continuous Sampling and Automatic Analysis for Silica in Modern High-Pressure Boilers" - Kenneth F. Schunk and T. Finnegan, Niagara Mohawk Power Corp., Buffalo, N. Y

"Application of the Forward Scattering Photometer to Aerosol Measurement" - David Sinclair, Johns-Manville Research Center, Manville, N. J.

"The Design of Automatic Trace Analyzers" -Henry J. Noebels, Beckman Scientific and Process Instruments Division, Fullerton, Calif.

"Some Recent Experiences on Feed Water Analysis on Board a Naval Vessel" - A. E. Gallant, U.S.S. *Tarawa*, Quonset Point Naval Air Station, Quonset Point, R. I.

PANEL DISCUSSION

PROBLEMS ON AUTOMATION OF CHEMICAL ANALYSIS AND INDUSTRIAL PROCESSES

Chairman: Jacques Kelly
Chas. Pfizer & Co., Inc., Brooklyn, N. Y.

Discussants:
T. Finnegan, Niagara Mohawk Power Corp., Buffalo, N. Y.
John B. Freeman, Bethlehem Steel Co., Bethlehem, Pa.
A. E. Gallant, U.S.S. *Tarawa,* Quonset Point Naval Air Station, Quonset Point, R. I.
Dwight Gillette, Refined Syrups and Sugars, Inc., Yonkers, N. Y.
Ira L. Shannon, School of Aviation Medicine, Air University, United States Air Force, Randolph AFB, Tex.
Leonard T. Skeggs, Western Reserve University, Cleveland, Ohio.
Edward Wagman, Head, Chemistry Section, Research and Development Division, Bureau of Ships, United States Navy, Washington, D. C.
William Nacorsky, Consolidated Electric Co. of New York, New York, N. Y.

Duncan A. Holaday
Chairman

Andres Ferrari
Vice-Chairman

NEW MEMBERS

Elected November 19, 1959

SUSTAINING MEMBERSHIP

Zizine, Louis A., Ph.D., Biology. Research Assistant, Laboratoire d'Endocrinologie, College de France, Paris, France.

ACTIVE MEMBERSHIP

Arrigo, Joseph J., B.S., Pharmaceutical Chemistry. Instructor, Research Staff, Kenmore Pharmacy Inc., Augusta, Me.

Atherton, Phyllis J., B.S., Physiology. Director of Laboratory, Genesee County Hospital, Flint, Mich.

Aufderheide, Arthur C., M.D., Laboratory Medicines. Pathologist, St. Mary's Hospital, Duluth, Minn.

Baldini, Mario, M.D., Hematology. Associate Director, Blood Research Laboratory, New England Center Hospital, Boston, Mass.

Ball, Roger A., D.V.M., Comparative Pathology. Research Fellow, University of Minnesota, College of Veterinary Medicine, St. Paul, Minn.

Bender, A. Douglas, M.S., Cardiovascular Physiology. Research Assistant, Lankenau Hospital, Philadelphia, Pa.

Block, Walter D., Ph.D., Dermatology Research. Associate Professor, Biological Chemistry, University Hospital, University of Michigan, Ann Arbor, Mich.

Bocher, Carol A., M.S., Endocrinology. Junior Biochemist, Smith Kline and French Laboratories, Philadelphia, Pa.

Christensen, John A., Ph.D., Tissue Culture. Director, Tissue Culture Department, St. Barnabas Medical Center, Newark, N. J.

DeWitt, Foster, M.D., Renal and Bone Metabolism and Pathology. Resident in Pathology, Kaiser Foundation Hospital, Oakland, Calif.

Dratman, Mary Bagan, M.D., Endocrinology. Associate, Woman's Medical College of Pennsylvania, Philadelphia, Pa.

Dressler, Robert, M.S., Mathematics and Physics. Executive Vice-President, Autometric Corporation, New York, N. Y.

Duke, Frederick Robert, Ph.D., Physical Chemistry. Professor, Chemistry, Iowa State University, Ames, Iowa.

Farmanfarmaian, Allahverdi, Ph.D., Zoology. Research Assistant, Hopkins Marine Station, Pacific Grove, Calif.

Feldmann, Edward G., Ph.D., Analytical Methods and Specifications for Drugs. Chairman-Elect and Associate Director of Revision, National Formulary, American Pharmaceutical Association, Washington, D.C.

Fox, Alfred Earl, B.S., Microbiology. Assistant Scientist, Warner-Lambert Research Institute, Morris Plains, N. J.

Gagliani, George Anthony, M.D., Geriatrics. Senior Physician, Commonwealth of Massachusetts, Department of Correction, Medfield, Mass.

Glaser, Philip, M.S., Medical Chemistry. Documentation Specialist, Philadelphia, Pa.

Greene, Marjorie H., M.D., Heart Disease. Private Practice, Hicksville, N. Y.

Hardiman, John Anthony, B.S., Applied Mathematics. Assistant Engineer, Sperry Gyroscope Company, Syosset, N. Y.

Harkin, James C., M.D., Pathology. Associate Director, Laboratories, The Hospital for Special Surgery, New York, N. Y.

Hill, Lucius D. III, M.D., Thoracic. Staff Surgeon, The Mason Clinic, Seattle, Wash.

Horn, Leif, Ph.D., Physiology. Assistant Professor, Pathology, Bellevue Medical Center, New York, N. Y.

Jareth, Mary, M.S., Microbiology. Instructor, Biology, Molloy Catholic College, Rockville Center, L. I., N. Y.

Kallir, John, M.A., Biochemistry. Vice-President, William D. McAdams, Inc., New York, N. Y.

Kelsey, R. Clifford, Ph.D., Endocrinology. Senior Research Scientist, Smith Kline and French Laboratories, Philadelphia, Pa.

Kritchman, Marilyn M., M.D., Anesthesiology. Director, Anesthesiology, St. Francis Hospital, Bronx, N. Y.

Krüger, Rodolfo Lopez, M.D., Cardiology-High Altitude Adaptation. Instructor, Medicine, University of California Medical Center, San Francisco, Calif.

Land, Edwin H., Sc.D., Physical Optics. Director, Research, Polaroid Corporation, Cambridge, Mass.

Lang, Raymond W., Ph.D., Microbiology. Assistant Professor, Graduate School, St. John's University, Jamaica, N. Y.

Levitsky, Leon R., M.D., Diabetes. Private Practice, Mt. Rainier, Md.

Levy, Robert C., M.D., Medicine. Private Practice, Chicago, Ill.

Lipscomb, Harry Shepherd, M.D., Endocrine Physiology. Assistant Professor, Physiology, Baylor University College of Medicine, Houston, Texas.

McBeth, Charles H., Ph.D., Biochemistry. Clinical Biochemist, Peninsula General Hospital, Salisbury, Md.

McGuire, James K., Ph.D., Climatology. United States Weather Bureau, New York, N. Y.

Marchisotto, Robert, Ph.D., Pharmaceutical Product Development. Group Leader, Pharmaceutical Research Section, Johnson and Johnson Research Center, New Brunswick, N. J.

Miller, Marjorie Ann., M.S., Biology. Instructor, Nursing, Teachers College, Columbia University, New York, N. Y.

Monse, Ernst Ulrich, Ph.D., Electromigration in Molten Salts. Assistant Research Specialist, Rutgers State University, Newark, N. J.

Moyers, Jack, M.D., Anesthesiology. Associate Professor of Medicine, State University of Iowa, Iowa City, Iowa.

Osborg, Hans, Ph.D., Lithium, Boron Hydrides. Vice-President and Technical Director, Chemirad Corporation, Port Washington, N. Y.

Peretz, Dwight I., M.D., Internal Medicine. Private Practice, West Vancouver, B.C., Canada.

Perlman, Jess, LL.B., Psychology and Psychiatry. Consulting Director, Grove School, Madison, Conn.

Perry, Gerald F., M.D., Neurology. Medical Director, Psychiatric Clinic, Mercy Hospital, Roslyn, N. Y.

Pruitt, John Crayton, M.D., Cancer. Medical Officer in Charge, Peripheral Blood Cytology Project, Baptist Hospital, Winston Salem, N. C.

Ramfjord, Sigurd P., Ph.D., Oral Pathology. Professor, Dentistry, University of Michigan School of Dentistry, Ann Arbor, Mich.

Ringertz, Nils Robert, M.K., Biology. Research Associate, Institute of Cell Research, Karolinska Institutet, Stockholm, Sweden.

Ross, Samuel D., Jr., A.B., Pharmacology. Pharmacologist, Smith Kline and French Laboratories, Philadelphia, Pa.

Schormuller, Anton, Ph.D., Organic Chemistry. Research Chemist, The Lubrizol Corporation, Wickliffe, Ohio.